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Serial No.: 09/591,789

Applicants: Marchalonis, J.J., et al.

Filing Date: 06/12/00

Priority Date: 06/12/00

Search Strategy

FILE 'USPATFULL' ENTERED AT 14:31:57 ON 09 FEB 2002

E MARCHALONIS J J/IN
L1 2 S E4
E WATSON RONALD R/IN
L2 3 S E3
E SCHLUTER SAMUEL F/IN
L3 3644 S (T-CELL RECEPTOR OR TCR)
L4 2225 S L3 AND (PEPTIDE? OR POLYPEPTIDE?)
L5 0 S L4 AND (CKPISGHNSLFWYRQT)
L6 241 S L4 AND (TH1 OR TH2 OR TH0)
L7 139 S L6 AND (HIV OR SIV OR FIV OR EIAV OR BIV OR CAEV)
L8 12 S L7 AND (TH1/CLM OR TH2/CLM OR TH0/CLM)
L9 289 S (TCR/CLM OR T-CELL RECEPTOR/CLM)
L10 185 S L9 AND L4
L11 94 S L10 AND (PEPTIDE/CLM OR POLYPEPTIDE/CLM)

FILE 'MEDLINE' ENTERED AT 14:44:05 ON 09 FEB 2002

E MARCHALONIS J J/AU
L12 237 S E3-E4
L13 3 S L12 AND (TH1 OR TH2 OR TH0)
L14 4 S L12 AND (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L15 4 S L14 NOT L13
L16 230 S L12 NOT (L13 OR L14)
E WATSON R R/AU
L17 246 S E3
L18 242 S L17 NOT L16
L19 17 S L18 AND (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L20 10 S L18 AND (TH1 OR TH2 OR TH0)
L21 10 S L20 NOT L19
E SCHLUTER S F/AU
L22 78 S E3
L23 10 S L22 NOT (L12 OR L17)

FILE 'WPIDS' ENTERED AT 15:01:07 ON 09 FEB 2002

E MARCHALONIS J J/IN
L24 3 S E3

FILE 'MEDLINE' ENTERED AT 15:03:40 ON 09 FEB 2002

L25 113498 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L26 461 S L25 AND (TH1 OR TH2 OR TH0)
L27 185 S L26 AND (MODULATION OR TREATMENT OR THERAP? OR REGULATION)
E FAUCI A/AU
L28 729 S E4
L29 2 S L28 AND (TH1 OR TH2 OR TH0)
L30 16 S L28 AND (IMMUNOPATHOGENESIS OR CYTOKINE DYSREGULATION)
L31 62 S L28 AND (IL-2 OR IL-4 OR IL-10)
L32 0 S L31 AND (TCR OR T-CELL RECEPTOR)
L33 11 S L28 AND (TCR OR T-CELL RECEPTOR)
L34 33 S L26 AND (TH0 OR TH0)
L35 626 S L25 AND (CYTOKINE THERAPY OR IMMUNOTHERAPY)
L36 77 S L35 AND (IL-2 OR INTERLEUKIN-2)

L1 ANSWER 1 OF 2 USPATFULL

1999:67011 T-cell receptor peptides and methods for preventing the progression to AIDS in an animal model.

Marchalonis, John J. , Tucson, AZ, United States

Watson, Ronald R., Tucson, AZ, United States

Dehghanpisheh, Keivan, Portland, OR, United States

Wang, Yuejian, St. Paul, MN, United States

Huang, Dennis S., Shaker Heights, OH, United States

Arizona Board of Regents on Behalf of the University of Arizona, Tucson,

AZ, United States (U.S. corporation)

US 5911990 19990615

APPLICATION: US 1996-696049 19960813 (8)

DOCUMENT TYPE: Utility; Granted.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A novel peptide from the T-cell receptor is shown to be effective in preventing the progression to AIDS in an animal model. Methods for delaying the progression to AIDS and restoring normal immunological responses in an animal model following infection are shown and comprise administering through various systemic routes T-cell receptor peptide V.beta. CDR1 to restore normal levels of Th1 cytokines interleukin 2 and interferon-.gamma., which are suppressed following infection, and those of Th2 derived cytokines interleukin 5, interleukin 6, interleukin 10, and immunoglobulin G, which are stimulated following infection.

CLM What is claimed is:
1. A method of modulating the immune response in a mammal infected with a C-type retrovirus or a lentivirus, comprising administering by a systemic route an amount of T-cell receptor V.beta. CDR1 peptide of SEQ ID NO:1 sufficient to stimulate the production of interleukin 2 and interferon-.gamma., and to suppress the production of interleukin 5, interleukin 6, interleukin 10, and immunoglobulin G.

2. The method of claim 1, wherein said lentivirus comprises HIV.

3. A method of altering the immune system response of a host infected with a C-type retrovirus or a lentivirus, comprising artificially introducing a T-cell receptor V.beta. CDR1 peptide of SEQ ID NO: 1 into the bloodstream or immune system by injection so as to artificially induce said immune system to stimulate production of Th1 cytokines or suppress production of Th2 derived cytokines.

4. The method of claim 3, wherein said lentivirus comprises HIV.

5. The method of claim 3, wherein said lentivirus comprises feline immunodeficiency virus.

6. A method of altering the immune system response of a host suffering from an infectious disease comprising artificially introducing a T-cell receptor V.beta. CDR1 peptide of SEQ ID NO: 1 into the bloodstream or immune system by injection so as to artificially induce said immune system to stimulate production of Th1 cytokines or suppress production of Th2 derived cytokines.

L15 ANSWER 1 OF 4 MEDLINE

1999326382 Document Number: 99326382. PubMed ID: 10398407. Recognition of defined epitopes by affinity-purified anti-immunoglobulin fab autoantibodies isolated from ***HIV*** -infected humans.
Marchalonis J J ; Garza A; Lake D F; Landsperger W J; Susal C. (Microbiology and Immunology, College of Medicine, Arizona Health Sciences Center, P.O. Box 24-5049, Tucson AZ 85724, USA.. dianah@u.arizona.edu) . JOURNAL OF MOLECULAR RECOGNITION, (1999 May-Jun) 12 (3) 169-76. Journal code: A00; 9004580. ISSN: 0952-3499. Pub. country: ENGLAND: United Kingdom. Language: English.

AB Infection of humans with ***HIV*** -1 has previously been independently shown to result in the generation of autoantibodies (AAbs) reactive with immunoglobulin Fab fragments (Heidelberg), and with autoantibodies to T-cell receptors (TCRs) (Tucson). Here, we carry out epitope mapping studies of affinity-purified AAbs to Fab fragments prepared from individual ***HIV*** -positive patients for their capacity to bind recombinant constructs and peptide-defined epitopes modeling TCR and Ig light chains. Some affinity-purified autoantibodies reacted strongly with TCRs expressed by intact T-cells, and recombinant Valpha/Vbeta constructs as well as with certain synthetic peptide epitopes. The binding reactions of affinity-purified AAbs of individual patients were distinct, and the AAb preparations consisted of populations of polyclonal lgs as reflected in specificity and isotype. AAb pools from individual patients all bound particular regions of TCR and Ig chains defined by comprehensive peptide synthesis including the CDR1 and Fr3 segments of the variable domains and the joining segment/switch peptide. In addition, other reactivities to restricted regions of alpha, beta and lambda light chains were documented. These results substantiate the cross-reactivity of TCR and Ig-Fab determinants, and are consistent with the hypothesis that autoantibodies arising as a consequence of ***HIV*** infection can have an immunomodulatory role.
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L15 ANSWER 2 OF 4 MEDLINE

97153482 Document Number: 97153482. PubMed ID: 9000486. Analysis of autoantibodies to T-cell receptors among ***HIV*** -infected individuals: epitope analysis and time course. ***Marchalonis J J*** ; Ampel N M; Schluter S F; Garza A; Lake D F; Galgiani J N; Landsperger W J. (College of Medicine, University of Arizona, Tucson, Arizona, 85724, USA.) CLINICAL IMMUNOLOGY AND IMMUNOPATHOLOGY, (1997 Feb) 82 (2) 174-89. Journal code: DEA; 0356637. ISSN: 0090-1229. Pub. country: United States. Language: English.

AB Individuals seropositive for ***human*** ***immunodeficiency*** ***virus*** type 1 (***HIV***) express elevated levels of autoantibodies (AAbs) directed against recombinant T-cell receptors (TCRs) and synthetic peptide epitopes duplicating beta chain markers. We performed longitudinal studies of anti-TCR AAbs in ***HIV*** -1-infected individuals, making comparisons with uninfected sera and sera from other individuals infected with a nonviral agent. We determined levels of autoantibodies by titration using enzyme-linked immunosorbent assay (ELISA) and developed a means for characterizing "autoantibody CDR recognition spectrotypes" for individual sera. Antibody levels against certain defined synthetic epitopes were substantially elevated in ***HIV*** -infected subjects relative to reactivities by control groups. Individual sera showed relatively high AAb levels to a subset of CDR1 peptide epitopes. Two patients who subsequently developed AIDS showed particular reactivity to Vbeta2.1, 8.1, 10.1, and 22.1 epitopes. Our results show that production AAbs to TCR Vbeta epitopes is a general

consequence of ***HIV*** infection. The response is individual but shows some restriction and shifts in AAb subpopulations often occur with time.

L15 ANSWER 3 OF 4 MEDLINE

96229155 Document Number: 96229155. PubMed ID: 8644504. Autoantibodies against peptide-defined epitopes of T-cell receptors in retrovirally infected humans and mice. ***Marchalonis J J*** ; Lake D F; Schluter S F; Dehghanpisheh K; Watson R R; Ampel N M; Galgiani J N. (University of Arizona, Tucson, USA.) ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1995) 383 211-22. Journal code: 2LU; 0121103. ISSN: 0065-2598. Pub. country: United States. Language: English.

AB Autoantibodies directed against peptide-defined epitopes of T-cell receptors occur in the serum of healthy humans with the levels and isotypic expression dependent upon physiological changes (aging, pregnancy) or upon the development of autoimmune disease. We carried out investigations of autoantibodies against Tcr peptide-defined epitopes in retroviral infections of humans (***HIV*** -1) and mice (LP-BM5 strain of murine leukemia virus) to determine whether infection with these agents disrupted the regulation of the production of these antibodies. Retroviral infection in humans resulted in increased levels of autoantibody production against putative immunoregulatory regions of the Tcr beta chain (V beta CDR1 and Fr3), a result reflecting a disruption of regulation. In addition, antigenic mimicry was observed with a cross-reaction shared between the common portion of the V3 neutralizing loop of the ***HIV*** -1 gp120 molecule and the joining segment of T-cell receptors (J beta). Infection of mice with the defective retrovirus resulted in the induction of antibodies directed particularly against V beta CDR1 peptide-defined determinants. Analysis of the virally induced response to a set of 8 CDR1 peptide epitopes indicated a selectivity to the process. It was possible to partially reverse aberrant cytokine changes correlated with the onset of murine MAIDS by administration of T-cell receptor peptides in saline. These results show that retroviral infection in humans and mice has a profound dysfunctional effect on the regulation of autoantibodies to T-cell receptors. The function of these autoantibodies in the immunopathogenesis of acquired immunodeficiency remains to be determined.

L15 ANSWER 4 OF 4 MEDLINE

95062162 Document Number: 95062162. PubMed ID: 7971973. Autoantibodies to the alpha/beta T-cell receptors in ***human***
immunodeficiency ***virus*** infection: dysregulation and mimicry. Lake D F; Schluter S F; Wang E; Bernstein R M; Edmundson A B; ***Marchalonis J J*** . (College of Medicine, University of Arizona, Tucson 85724.) PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1994 Nov 8) 91 (23) 10849-53. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States. Language: English.

AB Autoimmune reactivity is a consequence of infection with ***human***
immunodeficiency ***virus*** (***HIV***). We studied serological cross-reactions of purified IgG from sera of
HIV -infected individuals by using nested sets of synthetic overlapping peptides duplicating the covalent structures of T-cell receptors (TCRs) and immunoglobulin light chains and report that two processes of autoantibody production occur. (i) IgG autoantibodies to putative regulatory variable domain CDR1 and FR3 epitopes (where CDR is complementarity-determining region and FR is framework region) are present in pooled IgG from ***HIV*** -infected individuals at levels 10-fold greater than that in pooled IgG from healthy humans. (ii) Anti-TCR autoimmunization involves antigenic mimicry between a conserved peptide

stretch of the major neutralizing V3 loop determinant of ***HIV*** -1 gp120 and the conserved FR4 segment of the TCR V beta. Affinity-purified antibodies to the synthetic V3 loop peptide bound to a recombinant single-chain TCR and to a synthetic TCR joining segment peptide containing the FR4 sequence. Conversely, affinity-purified autoantibodies from pooled IgG from ***HIV*** -infected individuals to the TCR peptide bound the V3 loop peptide and a single-chain TCR. Inhibition studies indicated that the cross-reactive immunizing antigen was the V3 loop. These results bear upon the impact of ***HIV*** infection on immune regulation and on the selection of peptides for vaccine development.

L21 ANSWER 9 OF 10 MEDLINE

95363196 Document Number: 95363196. PubMed ID: 7636274. T cell receptor V beta complementarity-determining region 1 peptide administration moderates immune dysfunction and cytokine dysregulation induced by murine retrovirus infection. ***Watson R R*** ; Wang J Y; Dehghanpisheh K; Huang D S; Wood S; Ardestani S K; Liang B; Marchalonis J J. (Department of Family and Community Medicine, University of Arizona College of Medicine, Tucson 85724, USA.) JOURNAL OF IMMUNOLOGY, (1995 Aug 15) 155 (4) 2282-91. Journal code: IFB; 2985117R. ISSN: 0022-1767. Pub. country: United States. Language: English.

AB Murine AIDS, induced by LP-BM5 murine leukemia retrovirus infection, causes a progressive and profound immunodeficiency in female C57Bl/6 mice. Previously, we reported that autoantibodies were elevated during the initiation phases of this murine retrovirus infection and bound peptide determinants corresponding to CDR1 of several TCR V beta-chains. Therefore, we designed studies to determine whether administration of a major autoimmunogenic TCR V beta CDR1 peptide before or after infection with LP-BM5 retrovirus would modulate retrovirus-induced dysregulation of T cell function. Administration of the TCR V beta CDR1 peptide before murine retrovirus infection significantly prevented its suppression of splenic NK cell activity, T and B cell proliferation, and monokine (IL-6 and TNF-alpha) and ***Th1*** cytokine (IL-2 and IFN-gamma) release by splenocytes, and inhibited retrovirus-induced elevation of ***Th2*** cytokine (IL-5 and IL-10). Similar data were obtained with peptide immunization 2 wk after murine retrovirus infection at 6 and 16 wk postinfection. However, delaying peptide immunization until severe suppression of T and B cell mitogenesis had occurred did not restore their functions. Immunization with TCR V beta peptide prevents development of retrovirus-induced immune dysfunction, which suggests a possible pathogenic role of autoreactive T cells as regulatory elements.

L27 ANSWER 184 OF 185 MEDLINE

93039657 Document Number: 93039657. PubMed ID: 1418612. T helper cell immune dysfunction in asymptomatic, ***HIV*** -1-seropositive individuals: the role of ***TH1*** - ***TH2*** cross-***regulation***. Shearer G M; Clerici M. (Experimental Immunology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Md.) CHEMICAL IMMUNOLOGY, (1992) 54 21-43. Ref: 116. Journal code: AF7; 9001090. ISSN: 1015-0145. Pub. country: Switzerland. Language: English.

L27 ANSWER 176 OF 185 MEDLINE

95151146 Document Number: 95151146. PubMed ID: 7848519. The ***Th1*** - ***Th2*** hypothesis of ***HIV*** infection: new insights. Clerici M; Shearer G M. (Cattedra di Immunologia, Universita degli Studi, Milano, Italy.) IMMUNOLOGY TODAY, (1994 Dec) 15 (12) 575-81. Ref: 104. Journal code: AEA; 8008346. ISSN: 0167-5699. Pub. country: ENGLAND: United Kingdom. Language: English.

AB In their earlier, much quoted, viewpoint article, Mario Clerici and Gene Shearer examined the role of T helper 1 (***Th1***)- and ***Th2*** -type responses in immune dysregulation associated with ***human*** ***immunodeficiency*** ***virus*** (***HIV***) infection. In this article, they consider the complications of a ***Th1*** - ***Th2*** model raised by the nomenclature, discuss the issue of cytokine production by non-T cells, and compare data obtained from T-cell clones with heterogeneous populations of leukocytes from patients. They define Th-cell responses and cytokine profiles as 'type 1' and 'type 2', and reemphasize the importance of strong cellular immune responses, along with the cytokines that augment and maintain such responses, in protective immunity against ***HIV*** infection and AIDS progression. Finally, they present a model of activation-induced, cytokine-modulated, programmed cell death as a major factor in the pathogenesis of ***HIV*** infection and AIDS.

L27 ANSWER 175 OF 185 MEDLINE
95232493 Document Number: 95232493. PubMed ID: 7716511. AIDS research. Cytokines move from the margins into the spotlight. Balter M. SCIENCE, (1995 Apr 14) 268 (5208) 205-6. Journal code: UJ7; 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L27 ANSWER 156 OF 185 MEDLINE
96285771 Document Number: 96285771. PubMed ID: 8724835. Cytokines in immune ***regulation*** /pathogenesis in ***HIV*** infection. Shearer G M; Clerici M; Sarin A; Berzofsky J A; Henkart P A. (National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, USA.) CIBA FOUNDATION SYMPOSIUM, (1995) 195 142-7; discussion 147-53. Ref: 44. Journal code: D7X; 0356636. ISSN: 0300-5208. Pub. country: Netherlands. Language: English.

AB Two hallmarks of immunopathogenesis in the progression of ***HIV*** -infected individuals to AIDS are the loss of T helper (Th) cell function in response to antigens and the critical reduction in CD4+ T cell numbers. It is probable that these two phenomena are related. We observed that: (1) the failure to detect antigen-stimulated Th cell responses in vitro correlates with increased pokeweed mitogen/staphylococcal enterotoxin B (P/S)-stimulated and antigen-stimulated T cell death; and (2) both of these events are similarly modulated by immunoregulatory cytokines. Interleukin 2 (IL-2) and IL-12 (***Th1*** -type cytokines), as well as antibodies to IL-4 and IL-10 (which are ***Th2*** -type cytokines) restore in vitro Th cell responses to recall antigens such as influenza virus and ***HIV*** envelope synthetic peptides (env). P/S-induced T cell death affects both CD4+ and CD8+ T cell subsets, whereas death induced by stimulation with env affects only CD4+ T cells. In both examples, ***Th1*** -type cytokines and antibodies to ***Th2*** -type cytokines protect against T cell death. In contrast, IL-4 and IL-10 do not protect against death, and anti-IL-12 antibody can enhance T cell death. Our findings indicate that the loss of Th cell function and increased T cell death seen in vitro are correlated, and that in vivo ***HIV*** infection gives rise to inappropriate cytokines resulting in immune dysfunction and immunopathogenesis.

L27 ANSWER 177 OF 185 MEDLINE
95125467 Document Number: 95125467. PubMed ID: 7824955. ***TH2*** downregulation of macrophage ***HIV*** -1 replication. Montaner L J; Gordon S. SCIENCE, (1995 Jan 27) 267 (5197) 538-9. Journal code: UJ7; 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L27 ANSWER 164 OF 185 MEDLINE
96074662 Document Number: 96074662. PubMed ID: 7479952. Effects of

TH1 and ***TH2*** cytokines on CD8+ cell response against
 human ***immunodeficiency*** ***virus*** : implications for
 long-term survival. Barker E; Mackewicz C E; Levy J A. (Cancer Research
 Institute, University of California, San Francisco 94143-0128, USA.)
 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF
 AMERICA, (1995 Nov 21) 92 (24) 11135-9. Journal code: PV3; 7505876. ISSN:
 0027-8424. Pub. country: United States. Language: English.

AB CD8+ cells from long-term survivors [LTS; infected with ***human***
 immunodeficiency ***virus*** (***HIV***) for 10 or more
 years and having CD4+ cell counts of > or = 500 cells per microliters]
 have a 3-fold greater ability to suppress ***HIV*** replication than
 do CD8+ cells from patients who have progressed to disease (progressors)
 during the same time period. A change in the pattern of cytokines produced
 in the host from those that typically favor cell-mediated immunity (T
 helper 1, ***TH1*** or type 1) to those that down-regulate it (T
 helper 2, ***TH2*** or type 2) was investigated as a cause of this
 reduced CD8+ cell anti- ***HIV*** function. ***Treatment*** of CD8+
 cells from LTS with the ***TH1*** cytokine interleukin (IL)-2 enhanced
 their anti- ***HIV*** activity, whereas exposure of these cells to
 TH2 cytokines IL-4 or IL-10 reduced their ability to suppress
 HIV replication and to produce IL-2. IL-2 could prevent and
 reverse the inhibitory effects of IL-4 and IL-10. Moreover, prolonged
 exposure of CD8+ cells from some progressors to IL-2 improved the ability
 of these cells to suppress ***HIV*** replication. These observations
 support previous findings suggesting that strong CD8+ cell responses play
 an important role in maintaining an asymptomatic state in ***HIV***
 infection. The data suggest that the loss of CD8+ cell suppression of
 HIV replication associated with disease progression results from a
 shift in cytokine production within the infected host from a ***TH1***
 to a ***TH2*** pattern. ***Modulation*** of these cytokines could
 provide benefit to ***HIV*** -infected individuals by improving their
 CD8+ cell anti- ***HIV*** activity.

L27 ANSWER 127 OF 185 MEDLINE
 97381156 Document Number: 97381156. PubMed ID: 9238512. Role of cellular
 immunity in protection against ***HIV*** infection. Rowland-Jones S;
 Tan R; McMichael A. (Institute of Molecular Medicine, John Radcliffe
 Hospital, Headington, Oxford, United Kingdom.) ADVANCES IN IMMUNOLOGY,
 (1997) 65 277-346. Ref: 381. Journal code: 2N9; 0370425. ISSN: 0065-2776.
 Pub. country: United States. Language: English.

L27 ANSWER 119 OF 185 MEDLINE
 1998131999 Document Number: 98131999. PubMed ID: 9472657. ***HIV***
 -induced IL-6/IL-10 dysregulation of CD4 cells is associated with
 defective B cell help and autoantibody formation against CD4 cells. Weimer
 R; Zipperle S; Daniel V; Zimmermann R; Schimpf K; Opelz G. (Institute of
 Immunology, University of Heidelberg, Germany.) CLINICAL AND EXPERIMENTAL
 IMMUNOLOGY, (1998 Jan) 111 (1) 20-9. Journal code: DD7; 0057202. ISSN:
 0009-9104. Pub. country: ENGLAND: United Kingdom. Language: English.

AB To analyse CD4 cell cytokine secretion and helper/suppressor function at a
 clonal level we established 446 CD4+ T cell clones (TCC) in four healthy
 controls, three ***HIV*** - haemophilia patients, four CDC II, III and
 four CDC IV patients. Spontaneous TCC secretion of ***Th1*** cytokines
 (IL-2, interferon-gamma (IFN-gamma)) and ***Th2*** cytokines (IL-4,
 IL-6, IL-10) was determined by ELISA. TCC helper and suppressor functions
 were tested in a pokeweed mitogen (PWM)-stimulated allogeneic co-culture
 system using a reverse haemolytic plaque assay for assessment of B cell
 responses. There was a significant association of TCC surface marker
 expression (Leu-8, CD45RA) with TCC IL-6 secretion in healthy controls (P

< 0.01), ***HIV*** - patients ($P < \text{or} = 0.001$) and CDC II,III patients ($P < \text{or} = 0.01$) but not in CDC IV patients. Likewise, TCC expression of Leu-8 and CD45RA was significantly associated with TCC suppressor function in healthy controls ($P < \text{or} = 0.0005$) but not in ***HIV*** -infected patients. A reduced TCC helper frequency ($< \text{or} = 10\%$ of TCC) and an enhanced TCC suppressor frequency ($> 80\%$ of TCC) were detected only in those ***HIV*** -infected patients who showed an excessively increased TCC IL-6 secretion ($> 70\%$ of TCC) together with a significantly diminished TCC IL-10 secretion ($< \text{or} = 10\%$ of TCC). CD4 cell autoantibodies also were found only in patients with this type of cytokine dysregulation. These data indicate that CD4 cell surface markers lose their functional relevance in ***HIV*** -infected patients. ***HIV*** -induced IL-6/IL-10 dysregulation of CD4+ T cells, i.e. the up- ***regulation*** of spontaneous IL-6 and down- ***regulation*** of spontaneous IL-10 secretion, appears to be involved in inducing CD4 helper defects and may promote autoantibody formation against CD4 cells.

L27 ANSWER 113 OF 185 MEDLINE
1998208295 Document Number: 98208295. PubMed ID: 9548515. Priming with IL-4 and IL-13 during ***HIV*** -1 infection restores in vitro IL-12 production by mononuclear cells of ***HIV*** -infected patients. Marshall J D; Robertson S E; Trinchieri G; Chehimi J. (The Wistar Institute of Anatomy and Biology, Philadelphia, PA 19104, USA.) JOURNAL OF IMMUNOLOGY, (1997 Dec 1) 159 (11) 5705-14. Journal code: IFB; 2985117R. ISSN: 0022-1767. Pub. country: United States. Language: English.

AB The production of proinflammatory cytokines can be regulated by several factors that exert activating or inhibitory effects. IL-4, IL-10, IL-13, TGF-beta, and PGE2 have demonstrated a very wide range of potent macrophage-deactivating activities and, specifically, down- ***regulation*** of the production of many proinflammatory monokines. IL-12 plays a key role during immune response by providing a link between natural resistance and adaptive immunity. We and others have previously shown an impairment in IL-12 production by PBMC from ***HIV*** -1-infected individuals in response to various stimuli, but defining the mechanism responsible remains elusive. In this study, we observed that pretreatment of PBMC from patients with IL-4 or IL-13 for 24 h primes the cells for enhanced production of IL-12 in response to Staphylococcus aureus, and almost completely restores their deficient IL-12 production when compared with healthy controls. Although this priming effect was completely abrogated by IL-10 and PGE2, IL-10 was produced equivalently by untreated and IL-4- or IL-13-pretreated PBMC from both patients and controls. Additionally, indomethacin, which shuts off PGE2 synthesis, and cAMP-blocking reagents failed to restore or enhance IL-12 production. The priming effect of IL-4 and IL-13 is at the transcription level for both p40 and p35 genes. This complete restoration of IL-12 production by ***Th2*** -associated cytokines was unexpected in light of the mutually antagonistic roles of IL-12 and IL-4 in promoting ***Th1*** or ***Th2*** immune responses.

L27 ANSWER 87 OF 185 MEDLINE
1999217474 Document Number: 99217474. PubMed ID: 10202824. Persistent alterations in T-cell repertoire, cytokine and chemokine receptor gene expression after 1 year of highly active antiretroviral ***therapy*** . Martinon F; Michelet C; Peguillet I; Taoufik Y; Lefebvre P; Goujard C; Guillet J G; Delfraissy J F; Lantz O. (ICGM-INSERM U445, Hopital Cochin, Paris, France.) AIDS, (1999 Feb 4) 13 (2) 185-94. Journal code: AID; 8710219. ISSN: 0269-9370. Pub. country: ENGLAND: United Kingdom. Language: English.

AB OBJECTIVES: To examine T-cell repertoire modifications, the evolution of

T-helper (TH)1/ ***TH2*** cytokine imbalance and modifications in chemokine receptor expression when the viral load is decreased by 2-3 log10 copies/ml under highly active antiretroviral ***therapy*** (HAART). DESIGN: Sixteen patients previously treated with zidovudine and lamivudine, with CD4 cells below 300 x 10(6)/l and viraemia above 30000 copies/ml were treated by saquinavir and ritonavir together with both reverse transcriptase (RT) inhibitors (ANRS 069 trial). T-cell repertoire, chemokine receptor and lymphokine expression were studied from peripheral blood mononuclear cells sampled at weeks 0, 24 and 48. METHODS: T-cell repertoire study was carried out using the Immunoscope method. Interleukin (IL)-12 receptor beta2, CC-chemokine receptor (CCR)-3, CXC-chemokine receptor-4 and CCR-5 expression in CD4+ cells was measured by kinetic quantitative PCR and IL-2, IL-4, IL-10, IL-13, interferon (IFN)-gamma were measured using a quantitative RT-PCR assay with homologous internal standards. RESULTS: Repertoire alterations were more frequent in CD4- than in CD4+ cells and persisted despite undetectable viraemia. Increased CCR-3 expression and spontaneous IFN-gamma as well as mitogenic induced IL-13 were observed at baseline and decreased slightly under HAART. CONCLUSION: The CD8+ cell repertoire alterations were profound, whereas the CD4+ cell alterations were moderate and both persisted unchanged under HAART. The ***TH1*** / ***TH2*** imbalance was more related to ***TH2*** over-expression than to ***TH1*** deficiency and persisted for at least 1 year under HAART.

L29 ANSWER 2 OF 2 MEDLINE

94294789 Document Number: 94294789. PubMed ID: 8023143. Lack of evidence for the dichotomy of ***TH1*** and ***TH2*** predominance in HIV-infected individuals. Graziosi C; Pantaleo G; Gantt K R; Fortin J P; Demarest J F; Cohen O J; Sekaly R P; ***Fauci A S***. (Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892.) SCIENCE, (1994 Jul 8) 265 (5169) 248-52. Journal code: UJ7; 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

AB A switch from a T helper 1 (***TH1***) cytokine phenotype to a ***TH2*** phenotype has been proposed as a critical element in the progression of human immunodeficiency virus (HIV) disease. Here, constitutive cytokine expression was analyzed in unfractionated and sorted cell populations isolated from peripheral blood and lymph nodes of HIV-infected individuals at different stages of disease. Expression of interleukin-2 (IL-2) and IL-4 was barely detectable (or undetectable) regardless of the stage of disease. CD8+ cells expressed large amounts of interferon gamma and IL-10, and the levels of these cytokines remained stably high throughout the course of infection. Furthermore, similar patterns of cytokine expression were observed after stimulation in vitro of purified CD4+ T cell populations obtained from HIV-infected individuals at different stages of disease. These results indicate that a switch from the ***TH1*** to the ***TH2*** cytokine phenotype does not occur during the progression of HIV disease.

L33 ANSWER 2 OF 11 MEDLINE

97420772 Document Number: 97420772. PubMed ID: 9275214. Evidence for rapid disappearance of initially expanded HIV-specific CD8+ T cell clones during primary HIV infection. Pantaleo G; Soudeyns H; Demarest J F; Vaccarezza M; Graziosi C; Paolucci S; Daucher M; Cohen O J; Denis F; Biddison W E; Sekaly R P; ***Fauci A S***. (Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892, USA.. Giuseppe.Pantaleo@chuv.hospvd.ch) . PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1997 Sep 2) 94 (18) 9848-53. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States.

Language: English.

AB Down-regulation of the initial burst of viremia during primary HIV infection is thought to be mediated predominantly by HIV-specific cytotoxic T lymphocytes, and the appearance of this response is associated with major perturbations of the ***T*** ***cell***
receptor repertoire. Changes in the ***T*** ***cell***
receptor repertoire of virus-specific cytotoxic T lymphocytes were analyzed in patients with primary infection to understand the failure of the cellular immune response to control viral spread and replication. This analysis demonstrated that a significant number of HIV-specific T cell clones involved in the primary immune response rapidly disappeared. The disappearance was not the result of mutations in the virus epitopes recognized by these clones. Evidence is provided that phenomena such as high-dose tolerance or clonal exhaustion might be involved in the disappearance of these monoclonally expanded HIV-specific cytotoxic T cell clones. These findings should provide insights into how HIV, and possibly other viruses, elude the host immune response during primary infection.

L33 ANSWER 5 OF 11 MEDLINE
96201239 Document Number: 96201239. PubMed ID: 8607594. Immunopathogenic mechanisms of HIV infection. ***Fauci A S*** ; Pantaleo G; Stanley S; Weissman D. (National Institutes of Health, Bethesda, Maryland, USA.)
ANNALS OF INTERNAL MEDICINE, (1996 Apr 1) 124 (7) 654-63. Ref: 56.
Journal code: 5A6; 0372351. ISSN: 0003-4819. Pub. country: United States.
Language: English.

AB A complex array of multiphasic and multifactorial immunopathogenic mechanisms are involved in the establishment and progression of human immunodeficiency virus (HIV) disease. After primary infection, acute viremia occurs with wide dissemination of HIV. During this early viremic phase, the virus is trapped within the processes of follicular dendritic cells in the germinal centers of lymphoid tissue. Also, during this phase of primary infection, some patients show major expansions of certain subsets of CD8+ T cells that are identified by the expression of a particular variable region of the beta chain of the ***T*** -
cell ***receptor***. These expansions are manifestations of responses to HIV that may be important in controlling the progression of HIV infection. In addition, inappropriate immune activation and elevated secretion of certain proinflammatory cytokines occur during HIV infection; these cytokines play a role in the regulation of HIV expression in the tissues. Infection of progenitor cells in bone marrow and the thymus contribute to the lack of regeneration of immunocompetent cells. Dendritic cells are involved in the initiation and propagation of HIV infection in CD4+ T cells. In studies of long-term nonprogressors - persons who have stable CD4+ T-cell counts and no HIV disease progression despite years of HIV infection - preserved lymph node architecture, low viral burden, and viral expression were found.

L31 ANSWER 3 OF 62 MEDLINE
1999432163 Document Number: 99432163. PubMed ID: 10500107. Latent reservoirs of HIV: obstacles to the eradication of virus. Chun T W;
Fauci A S. (Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892, USA.. twchun@nih.gov) . PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1999 Sep 28) 96 (20) 10958-61.
Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States.
Language: English.

AB The use of highly active antiretroviral therapy (HAART) in the treatment of HIV-1-infected individuals has provided a considerable amount of

information regarding the dynamics of viral replication and has resulted in enormous advances in HIV therapeutics. The profound suppression of plasma viremia in HIV-infected individuals receiving HAART has resulted in a highly beneficial clinical effect and a dramatic decrease in the death rate attributable to AIDS. Nonetheless, the persistence of reservoirs of HIV, including latently infected, resting CD4+ T cells that can give rise to infectious HIV upon stimulation in vitro, has posed a sobering challenge to the long-term control or eradication of HIV in infected individuals receiving HAART. Although a recent study has demonstrated that the size of the pool of latently infected, resting CD4+ T cells can be markedly diminished with intermittent interleukin (***IL*** - ***2***) and continuous HAART, complete eradication of HIV in infected individuals remains extremely problematic. Here we discuss recent developments in studies of the latent reservoir of HIV in patients receiving HAART and implications for the long-term treatment of infected individuals and eradication of the infection.

L31 ANSWER 4 OF 62 MEDLINE

1999297913 Document Number: 99297913. PubMed ID: 10371503. Effect of interleukin-2 on the pool of latently infected, resting CD4+ T cells in HIV-1-infected patients receiving highly active anti-retroviral therapy. Chun T W; Engel D; Mizell S B; Hallahan C W; Fischette M; Park S; Davey R T Jr; Dybul M; Kovacs J A; Metcalf J A; Mican J M; Berrey M M; Corey L; Lane H C; ***Fauci A S*** . (Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland 20892, USA.. twchun@nih.gov) . NATURE MEDICINE, (1999 Jun) 5 (6) 651-5. Journal code: CG5; 9502015. ISSN: 1078-8956. Pub. country: United States. Language: English.

AB The size of the pool of resting CD4+ T cells containing replication-competent HIV in the blood of patients receiving intermittent interleukin (***IL***)- ***2*** plus highly active anti-retroviral therapy (HAART) was significantly lower than that of patients receiving HAART alone. Virus could not be isolated from the peripheral blood CD4+ T cells in three patients receiving ***IL*** - ***2*** plus HAART, despite the fact that large numbers of resting CD4+ T cells were cultured. Lymph node biopsies were done in two of these three patients and virus could not be isolated. These results indicate that the intermittent administration of ***IL*** - ***2*** with continuous HAART may lead to a substantial reduction in the pool of resting CD4+ T cells that contain replication-competent HIV.

L31 ANSWER 5 OF 62 MEDLINE

1998317020 Document Number: 98317020. PubMed ID: 9653086. Induction of HIV-1 replication in latently infected CD4+ T cells using a combination of cytokines. Chun T W; Engel D; Mizell S B; Ehler L A; ***Fauci A S*** . (Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland 20892, USA.. twchun@nih.gov) . JOURNAL OF EXPERIMENTAL MEDICINE, (1998 Jul 6) 188 (1) 83-91. Journal code: I2V; 2985109R. ISSN: 0022-1007. Pub. country: United States. Language: English.

AB Although it has been demonstrated that certain cytokines, particularly proinflammatory cytokines, can enhance ongoing viral replication in peripheral blood mononuclear cells (PBMCs) of HIV-1-infected individuals, it is unclear what role these cytokines play in the induction of HIV-1 replication in latently infected, resting CD4(+) T cells. This study demonstrates that the in vitro combination of the proinflammatory cytokines interleukin (IL)-6 and tumor necrosis factor (TNF)-alpha together with the immunoregulatory cytokine ***IL*** - ***2*** are potent inducers of viral replication in highly purified, latently

infected, resting CD4+ T cells derived from HIV-infected individuals who are antiretroviral therapy-naïve as well as those who are receiving highly active antiretroviral therapy (HAART). Viral replication induced by this combination of cytokines was completely suppressed in the presence of HAART in vitro. Given that an array of cytokines, including IL-6, TNF-alpha, and ***IL** - ***2***, are copiously expressed in the microenvironment of the lymphoid tissues, which harbor the latent viral reservoirs, induction of HIV by this combination of cytokines may in part explain the commonly observed reappearance of detectable plasma viremia in HIV-infected individuals in whom HAART was discontinued. Moreover, since it is likely that these infected cells die upon activation of virus and that HAART prevents spread of virus to adjacent cells, the observation that this combination of cytokines can markedly induce viral replication in this reservoir may have important implications for the activation-mediated diminution of the latent reservoir of HIV in patients receiving HAART.

L31 ANSWER 13 OF 62 MEDLINE
96320760 Document Number: 96320760. PubMed ID: 8739561. Interleukin-2 and human immunodeficiency virus infection: pathogenic mechanisms and potential for immunologic enhancement. Kinter A; ***Fauci A S***. (Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Md, USA.) IMMUNOLOGIC RESEARCH, (1996) 15 (1) 1-15. Ref: 136. Journal code: IMR; 8611087. ISSN: 0257-277X. Pub. country: United States. Language: English.

AB A hallmark of human immunodeficiency virus (HIV) infection is the progressive loss of CD4+ T lymphocytes; however, qualitative defects in immune responses occur prior to the precipitous drop CD4+ T cell numbers. One of the first immunologic defects to be described in HIV-infected individuals is a deficiency in interleukin (***IL**)- ***2*** production. The addition of ***IL** - ***2*** in vitro to cultures of mononuclear cells from HIV-infected individuals partially or completely restored certain defective cellular immune responses. However, production of or addition of ***IL** - ***2*** has also been associated with increased viral replication in infected T cells. These observations underscore the pernicious correlation between immune activation and HIV replication. However, recent in vitro and in vivo studies have provided promising preliminary results suggesting that, at least at certain stages of disease, the benefits of ***IL** - ***2*** mediated immune enhancement may outweigh or override the inductive effects of this cytokine on HIV production.

L31 ANSWER 14 OF 62 MEDLINE
96210653 Document Number: 96210653. PubMed ID: 8633076. Kinetics of cytokine expression during primary human immunodeficiency virus type 1 infection. Graziosi C; Gantt K R; Vaccarezza M; Demarest J F; Daucher M; Saag M S; Shaw G M; Quinn T C; Cohen O J; Welbon C C; Pantaleo G; ***Fauci A S***. (Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892-1876, USA.) PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996 Apr 30) 93 (9) 4386-91. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States. Language: English.

AB In the present study, we have determined the kinetics of constitutive expression of a panel of cytokines [interleukin (***IL**) ***2***, ***IL** - ***4***, IL-6, ***IL** - ***10***, interferon gamma (IFN-gamma), and tumor necrosis factor alpha (TNF-alpha)] in sequential peripheral blood mononuclear cell samples from nine individuals with primary human immunodeficiency virus infection. Expression of

IL - ***2*** and ***IL*** - ***4*** was barely detected in peripheral blood mononuclear cells. However, substantial levels of ***IL*** - ***2*** expression were found in mononuclear cells isolated from lymph node. Expression of IL-6 was detected in only three of nine patients, and IL-6 expression was observed when transition from the acute to the chronic phase had already occurred. Expression of ***IL*** - ***10*** and TNF-alpha was consistently observed in all patients tested, and levels of both cytokines were either stable or progressively increased over time. Similar to ***IL*** - ***10*** and TNF-alpha, IFN-gamma expression was detected in all patients; however, in five of nine patients, IFN-gamma expression peaked very early during primary infection. The early peak in IFN-gamma expression coincided with oligoclonal expansions of CD8+ T cells in five of six patients, and CD8+ T cells mostly accounted for the expression of this cytokine. These results indicate that high levels of expression of proinflammatory cytokines are associated with primary infection and that the cytokine response during this phase of infection is strongly influenced by oligoclonal expansions of CD8+ T cells.

L31 ANSWER 16 OF 62 MEDLINE
95353764 Document Number: 95353764. PubMed ID: 7627621. ***IL*** - ***10*** synergizes with multiple cytokines in enhancing HIV production in cells of monocytic lineage. Weissman D; Poli G; ***Fauci A S*** . (Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892, USA.) JOURNAL OF ACQUIRED IMMUNE DEFICIENCY SYNDROMES AND HUMAN RETROVIROLOGY, (1995 Aug 15) 9 (5) 442-9. Journal code: B7J; 9501482. ISSN: 1077-9450. Pub. country: United States. Language: English.

AB Several cytokines, whose expression is increased in human immunodeficiency virus (HIV)-infected individuals, can enhance virus replication in CD4+ T lymphocytes and mononuclear phagocytes (MP). We have previously reported that interleukin (***IL***)- ***10*** inhibited HIV replication in acutely infected monocyte-derived macrophages (MDM) at concentrations that completely blocked the production of endogenous tumor necrosis factor-alpha (TNF-alpha) and IL-6 from infected cells. In the present study, lower concentrations of ***IL*** - ***10*** , which were unable to completely suppress endogenous cytokines, paradoxically enhanced HIV replication in MDM induced by other cytokines. This synergistic induction of HIV expression by ***IL*** - ***10*** in combination with TNF-alpha, IL-6, and other cytokines was also observed in the chronically infected promonocytic cell line, U1. The enhancing effect of ***IL*** - ***10*** was correlated with an increase in HIV mRNA accumulation and potentiation of phorbol ester-induced long terminal repeat-driven transcription that was independent of the NF-kappa B and Sp1 transcription factors. Thus, ***IL*** - ***10*** is a cytokine capable of exerting complex regulatory effects on HIV expression in MP as a function of its own concentration and of the presence of other HIV regulatory cytokines.

L31 ANSWER 18 OF 62 MEDLINE
95151356 Document Number: 95151356. PubMed ID: 7848677. Interleukin 10 blocks HIV replication in macrophages by inhibiting the autocrine loop of tumor necrosis factor alpha and interleukin 6 induction of virus. Weissman D; Poli G; ***Fauci A S*** . (Laboratory of Immunoregulation, National Institutes of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland 20892.) AIDS RESEARCH AND HUMAN RETROVIRUSES, (1994 Oct) 10 (10) 1199-206. Journal code: ART; 8709376. ISSN: 0889-2229. Pub. country: United States. Language: English.

AB Human interleukin 10 is a pleiotropic cytokine capable of suppressing

cytokine production from macrophages and T cells; in addition, it exerts complex regulatory effects on CD8+ T cells, natural killer cells, vascular endothelial cells, and B lymphocytes. Levels of ***IL*** - ***10*** are elevated in HIV-infected individuals, suggesting that this cytokine may play a role in the suppression of T cell and monocyte/macrophage function typical of HIV disease. In this article, ***IL*** - ***10*** blocked HIV-induced tumor necrosis factor alpha and interleukin 6 secretion and inhibited HIV replication in monocyte-derived macrophages (MDMs). The inhibition by ***IL*** - ***10*** was correlated with a block in endogenous TNF-alpha and IL-6 secretion from HIV-infected MDMs.

L34 ANSWER 32 OF 33 MEDLINE

94294788 Document Number: 94294788. PubMed ID: 8023142. Ability of ***HIV*** to promote a ***TH1*** to ***TH0*** shift and to replicate preferentially in ***TH2*** and ***TH0*** cells. Maggi E; Mazzetti M; Ravina A; Annunziato F; de Carli M; Piccinni M P; Manetti R; Carbonari M; Pesce A M; del Prete G; +. (Division of Clinical Immunology and Allergy, University of Florence, Italy.) SCIENCE, (1994 Jul 8) 265 (5169) 244-8. Journal code: UJ7; 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

AB Both interferon gamma (IFN-gamma) produced by T helper 1 (***TH1***) lymphocytes and interleukin-4 (IL-4) produced by ***TH2*** lymphocytes were reduced in either bulk circulating mononuclear cells or mitogen-induced CD4+ T cell clones from the peripheral blood of individuals infected with ***human*** ***immunodeficiency*** ***virus*** (***HIV***). There was a preferential reduction in clones producing IL-4 and IL-5 in the advanced phases of infection. However, enhanced proportions of CD4+ T cell clones producing both ***TH1*** -type and ***TH2*** -type cytokines (***TH0*** clones) were generated from either skin-infiltrating T cells that had been activated in vivo or peripheral blood T cells stimulated by antigen in vitro when cells were isolated from ***HIV*** -infected individuals. All ***TH2*** and most ***TH0*** clones supported viral replication, although viral replication was not detected in any of the ***TH1*** clones infected in vitro with ***HIV*** . These results suggest that ***HIV*** (i) does not induce a definite ***TH1*** to ***TH2*** switch, but can favor a shift to the ***TH0*** phenotype in response to recall antigens, and (ii) preferentially replicates in CD4+ T cells producing ***TH2*** -type cytokines (***TH2*** and ***TH0***).

L34 ANSWER 31 OF 33 MEDLINE

95000921 Document Number: 95000921. PubMed ID: 7917511. An alternative view of the ***Th1*** / ***Th2*** switch hypothesis in ***HIV*** infection. Romagnani S; Maggi E; Del Prete G. (Division of Allergology and Clinical Immunology, Istituto di Clinica Medica III, University of Florence, Italy.) AIDS RESEARCH AND HUMAN RETROVIRUSES, (1994 May) 10 (5) iii-ix. Ref: 49. Journal code: ART; 8709376. ISSN: 0889-2229. Pub. country: United States. Language: English.

AB A theory that seeks to explain what induces the relentless progression of ***HIV*** -infected subjects to AIDS has received considerable attention. This theory holds that ***HIV*** infection results in a ***Th1*** / ***Th2*** switch. However, analysis of constitutive cytokine mRNA expression in lymphoid tissues from ***HIV*** -infected individuals did not confirm an in vivo ***Th1*** / ***Th2*** switch. Moreover, data available at the level of in vitro-stimulated peripheral blood mononuclear cells or cloned T cells do not provide clear evidence for a definite switch to the ***Th2*** responses in any ***HIV*** -infected subject and in any phase of ***HIV*** infection. At most, currently

available data on the profile of cytokines released in response to in vitro stimulation suggest a ***Th1*** -to- ***Th0*** shift in a proportion of memory CD4+ T cells. On the other hand, experiments of in vitro infection with ***HIV*** of already established CD4+ T cell clones indicated that ***Th2*** and ***Th0*** cells support ***HIV*** replication better than ***Th1*** cells, suggesting that early destruction of ***Th2*** cells by direct or indirect ***HIV*** -mediated cell killing may occur. Finally, in some ***HIV*** -infected individuals with low CD4+ T cell counts, a prevalence of CD8+ T cells producing type 2 cytokines was found in both peripheral blood and skin. Thus, although the induction of a general ***Th2*** state in ***HIV*** infection is not proven, enhanced production of type 2 cytokines may occur in a proportion of ***HIV*** -infected individuals and play some role in the pathogenesis of the disease.

L34 ANSWER 29 OF 33 MEDLINE

95122085 Document Number: 95122085. PubMed ID: 7821929. Role of ***TH1*** / ***TH2*** cytokines in ***HIV*** infection. Romagnani S; Del Prete G; Manetti R; Ravina A; Annunziato F; De Carli M; Mazzetti M; Piccinini M P; D'Elia M M; Parronchi P; +. (Department of Allergy & Clinical Immunology, University of Florence, Italy.) IMMUNOLOGICAL REVIEWS, (1994 Aug) 140 73-92. Ref: 65. Journal code: GG4; 7702118. ISSN: 0105-2896. Pub. country: Denmark. Language: English.

AB Different experimental approaches were used to prove or disprove the " ***TH1*** / ***TH2*** switch theory" of ***HIV*** -infection. No increase, or even a decrease, in the production of ***TH2*** -type cytokines (IL-4, IL-5, and IL-10) by either bulk circulating mononuclear cells or CD4+ T-cell clones generated by PHA stimulation of single T cells from ***HIV*** -infected individuals in all stages of disease compared to ***HIV*** -negative donors was observed. However, enhanced proportions of CD4+ T-cell clones able to produce both ***TH1*** -type and ***TH2*** -type cytokines (***TH0*** clones) were derived from either skin-infiltrating, in vivo-activated, T cells or in vitro antigen-stimulated peripheral blood T cells of ***HIV*** -infected individuals. Of note, ***TH1*** , ***TH2*** and ***TH0*** clones obtained from ***HIV*** -seronegative healthy donors showed different ability to support viral replication after infection with ***HIV*** in vitro. All ***TH2*** and most ***TH0*** clones supported ***HIV*** replication efficiently, whereas ***TH1*** clones did not. These results suggest preferential ***HIV*** replication in T cells producing ***TH2*** -type cytokines rather than ***TH1*** / ***TH2*** switch in ***HIV*** infection.

L34 ANSWER 22 OF 33 MEDLINE

96332554 Document Number: 96332554. PubMed ID: 8706325. CD8+ lymphocyte phenotype and cytokine production in long-term non-progressor and in progressor patients with ***HIV*** -1 infection. Zanussi S; Simonelli C; D'Andrea M; Caffau C; Clerici M; Tirelli U; DePaoli P. (Department of Microbiology-Immunology-Virology, C.R.O. Aviano, Italy.) CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1996 Aug) 105 (2) 220-4. Journal code: DD7; 0057202. ISSN: 0009-9104. Pub. country: ENGLAND: United Kingdom. Language: English.

AB In most ***HIV*** -1-infected patients, clinical and immunological progression develops within a few years. Few infected people, termed long-term non-progressors (LTNP), remain healthy and immunologically stable for a long time. The factors governing the maintenance of this condition are not well known, but it is conceivable that CD8+ lymphocytes, cells that play a central role in controlling in vitro ***HIV*** replication, may have a part in vivo in this process. The aim of this

study was to characterize the phenotypic profile and the cytokine production of CD8+ cells in a group of LTNP patients who had stable CD4+ cell counts (> 500/mm3) for at least 7 years. Their CD8+ absolute numbers were similar to a control group composed of ***HIV*** -1+ patients who have a progressive decline of their CD4+ cell counts. However, our multiparameter immunofluorescence studies show that a clinical and immunologically stable condition is associated with the presence of a CD28+, CD95 strongly positive CD8+ population, while disease progression is marked by the CD28-CD95+CD8+ subset. Purified CD8+ cells from LTNP retain their ability to produce IL-2, interferon-gamma (IFN-gamma) and, to a lesser degree, to produce IL-10 and IL-4. In contrast, CD8+ cells from progressors are unable to secrete IL-2 and IL-10. Although CD8+ cytokine profile does not fit with the proposed T helper (Th)1/ ***Th2*** switch in progressive ***HIV*** infection, LTNP CD8+ T cells maintain their capacity to produce IL-2 and IL-10 (***Th0*** -like), a pattern very similar to that observed in normal ***HIV*** healthy controls. We suggest that CD8+ cells expressing CD28, CD95 and having a ***Th0*** -like profile may be considered to be associated with long-term survival.

L36 ANSWER 4 OF 77 MEDLINE

2001469289 Document Number: 21405432. PubMed ID: 11514956. Different rates of CD4+ and CD8+ T-cell proliferation in ***interleukin*** - ***2*** -treated ***human*** ***immunodeficiency*** ***virus*** -positive subjects. Caggiari L; Zanussi S; Crepaldi C; Bortolin M T; Caffau C; D'Andrea M; De Paoli P. (Division of Microbiology, Immunology and Virology, Centro di Riferimento Oncologico, IRCCS, 33081 Aviano, Italy.) CYTOMETRY, (2001 Aug 15) 46 (4) 233-7. Journal code: D92; 8102328. ISSN: 0196-4763. Pub. country: United States. Language: English.

AB BACKGROUND: ***Interleukin*** - ***2*** (***IL*** - ***2***) has been used successfully to increase CD4 cell counts in patients who are ***human*** ***immunodeficiency*** ***virus*** (***HIV***) positive. The mechanisms involved in this phenomenon are unknown. We hypothesized that a differential proliferation rate of CD4+ compared with CD8+ lymphocytes could be related to the increase of CD4 counts and of CD4/CD8 ratios that occur in ***HIV*** + patients during ***IL*** - ***2*** treatment. METHODS: We enrolled in our study 14 ***HIV*** + patients treated with ***IL*** - ***2*** or with highly active antiretroviral therapy (HAART) during a 96-week observation period. Using flow cytometry, we measured longitudinally the expression of the Ki67 antigen in peripheral blood CD4+ and CD8+ lymphocyte subsets. RESULTS: Compared with HAART alone, ***IL*** - ***2*** produced a rapid increase of Ki67+ proliferating CD4 cells and a concomitant increase of the CD4/CD8 ratios, whereas the corresponding CD8 proliferation increased slightly. On the contrary, HAART alone was effective in suppressing equally both CD4 and CD8 proliferation. CONCLUSIONS: Our results suggest a selective activity of ***IL*** - ***2*** on CD4 T-cell proliferation; on the contrary, CD8-specific proliferation is affected minimally during treatment. This information may offer the potential to plan correctly immune activating regimens. Copyright 2001 Wiley-Liss, Inc.

L36 ANSWER 6 OF 77 MEDLINE

2001414990 Document Number: 21357617. PubMed ID: 11465092. ***Interleukin*** - ***2*** induced immune effects in ***human*** ***immunodeficiency*** ***virus*** -infected patients receiving intermittent ***interleukin*** - ***2*** ***immunotherapy***. Kovacs J A; Vogel S; Metcalf J A; Baseler M; Stevens R; Adelsberger J; Lempicki R; Hengel R L; Sereti I; Lambert L; Dewar R L; Davey R T Jr; Walker R E; Falloon J; Polis M A; Masur H; Lane H C. (Critical Care Medicine Department, Clinical Center, National Institutes of Health,

Bethesda, USA.. jkovacs@nih.gov) . EUROPEAN JOURNAL OF IMMUNOLOGY, (2001 May) 31 (5) 1351-60. Journal code: EN5; 1273201. ISSN: 0014-2980. Pub. country: Germany: Germany, Federal Republic of. Language: English.

AB To characterize the immunological effects of intermittent ***IL*** - ***2*** therapy, which leads to selective increases in CD4+ T lymphocytes in ***HIV*** -infected patients, 11 patients underwent extensive immunological evaluation. While ***IL*** - ***2*** induced changes in both CD4+ and CD8+ cell number acutely, only CD4+ cells showed sustained increases following discontinuation of ***IL*** - ***2*** . Transient increases in expression of the activation markers CD38 and HLA-DR were seen on both CD4+ and CD8+ cells, but CD25 (a chain of the ***IL*** - ***2*** receptor) increased exclusively on CD4+ cells. This increase in CD25 expression was sustained for months following discontinuation of ***IL*** - ***2*** , and was seen in naive as well as memory cells. ***IL*** - ***2*** induced cell proliferation, but tachyphylaxis to these proliferative effects developed after 1 week despite continued ***IL*** - ***2*** administration. It thus appears that sustained CD25 expression selectively on CD4+ cells is a critical component of the immunological response to ***IL*** - ***2*** , and that intermittent administration of ***IL*** - ***2*** is necessary to overcome the tachyphylaxis to ***IL*** - ***2*** -induced proliferation.

L36 ANSWER 10 OF 77 MEDLINE

2001363436 Document Number: 21317671. PubMed ID: 11424974. Low-dose daily ***interleukin*** - ***2*** ***immunotherapy*** : accelerating immune restoration and expanding ***HIV*** -specific T-cell immunity without toxicity. Smith K A. (Division of Immunology, Weill Medical College of Cornell University, New York, New York 10021, USA.. kasmith@med.cornell.edu) . AIDS, (2001 Feb) 15 Suppl 2 S28-35. Ref: 49. Journal code: AID; 8710219. ISSN: 0269-9370. Pub. country: England: United Kingdom. Language: English.

AB There is now a great deal of interest in therapies focused on improving the function of the immune system in the treatment of individuals infected with the ***HIV*** . Although the antiviral drugs effectively suppress replication of the virus, they cannot cure the infection. Therefore, it now appears that both antivirals and immune system stimulants will be necessary to maximally suppress residual latent virus, thereby allowing the discontinuation of the antivirals without relapse of detectable plasma virus. ***Interleukin*** - ***2*** (***IL*** - ***2***) the first cytokine to be discovered at the molecular level has been used as a therapeutic in ***HIV*** infection, because it is critical for a normal functioning immune response. ***IL*** - ***2*** is essential for the survival and proliferative expansion of antigen-activated T cells and natural killer (NK) cells, and also for promoting their differentiated functions of cytokine secretion and cytolysis. However, as ***IL*** - ***2*** stimulates both the innate and acquired immune responses, when used as a therapeutic it can lead to severe toxicity when given in high doses. This review focuses on low dose, daily ***IL*** - ***2*** therapy, used to accelerate the recovery of the immune system when viral replication is suppressed maximally with antivirals. In addition, the principles of the use of ***IL*** - ***2*** to activate ***HIV*** -specific immune reactivity are discussed. At least two signals are required to promote the proliferative expansion and function of antiviral effector lymphocytes, ***HIV*** antigens and ***IL*** - ***2*** .

L36 ANSWER 11 OF 77 MEDLINE

2001317070 Document Number: 21285055. PubMed ID: 11392678. Improving immune function and controlling viral replication in ***HIV***

-1-infected patients with immune-based therapies. Angel J B. (Division of Infectious Diseases, Ottawa Hospital-General Campus, University of Ottawa, Ottawa.) AIDS Read, (2001 Apr) 11 (4) 209-21. Ref: 85. Journal code: DL5; 9206753. ISSN: 1053-0894. Pub. country: United States. Language: English.

AB Restoring and preserving immune function is a key component to successfully managing ***HIV*** -1 disease. Phase II/III studies have evaluated the safety and immunologic effects of immune-based therapies, including granulocyte-macrophage colony-stimulating factor, ***interleukin*** - ***2*** , and an inactivated ***HIV*** -1 immunogen, as adjuncts to antiretroviral therapy. Addition of each of these immune-based therapies to a background antiretroviral regimen enhanced, to varying degrees, immunologic function and suppression of viral replication in ***HIV*** -1-infected patients, suggesting a potential role for immune-based therapies in the treatment of ***HIV*** -1 disease. Further studies are needed to better characterize specific immunologic and virologic effects in different patient populations and to determine their impact on clinical outcomes.

L4 ANSWER 18 OF 20 MEDLINE

92194934 Document Number: 92194934. PubMed ID: 1347855. ***T*** -
cell ***receptor*** variable gene products and early
HIV -1 infection. Dalglish A G; Wilson S; Gompels M; Ludlam C; Gazzard B; Coates A M; Habeshaw J. (Department of Cellular and Molecular Sciences, St George's Hospital Medical School, London, UK.) LANCET, (1992 Apr 4) 339 (8797) 824-8. Journal code: L0S; 2985213R. ISSN: 0140-6736. Pub. country: ENGLAND: United Kingdom. Language: English.

AB To assess the hypothesis that the ***human*** ***immunodeficiency***
virus (***HIV***) might mimic major histocompatibility complex (MHC) allodeterminants and interact with T-cell receptors (TCRs) of alloreactive T-cells, we have done a preliminary analysis of the range of alpha beta ***TCR*** gene products in 16 ***HIV*** -1-seropositive individuals with normal CD4 counts and in 16 healthy ***HIV*** -1-negative controls. Using a panel of monoclonal ***antibodies*** with a two-colour direct immunofluorescence method, we found a significant increase in the expression of the V beta 5.3 subfamily in the ***HIV*** -positive patient group compared with controls (p less than 0.01). Selected increase in expression of V beta sequences has been described in various autoimmune conditions and our findings raise the possibility that the ***immunopathological*** damage from ***HIV*** infection may be due to the induction of autoreactivity. If ***HIV*** does mimic MHC II, the normal immune response to the virus could represent an autoimmune process similar to graft-versus-host disease.

L4 ANSWER 16 OF 20 MEDLINE

93236722 Document Number: 93236722. PubMed ID: 8476511. Changes in the cytotoxic T-cell repertoire of ***HIV*** -1-infected individuals: relationship to disease progression. Grant M D; Smaill F M; Laurie K; Rosenthal K L. (Department of Pathology, McMaster University Health Sciences Centre, Hamilton, Ontario, Canada.) VIRAL IMMUNOLOGY, (1993 Spring) 6 (1) 85-95. Journal code: AD0; 8801552. ISSN: 0882-8245. Pub. country: United States. Language: English.

AB The repertoire of antigen-specific receptors expressed on T lymphocytes is shaped by fixed genetic and variable environmental selective pressures. Recent technological advances have enabled the analysis of ***T*** -
cell ***receptor*** (***TCR***) expression in the context of selective pressures arising through normal immune system development and also through pathological features of disease. The pathological

features of acquired immune deficiency syndrome (AIDS) are reflected by selective depletion of particular T lymphocyte subsets and expansion of others. An important question concerning the ***immunopathogenesis*** of AIDS is whether or not the perturbation of the CD4+ and CD8+ T-cell subsets following infection with ***human*** ***immunodeficiency*** ***virus*** (***HIV***) is selective based on ***TCR*** variable (V) region gene expression. To address this question, we have functionally analyzed ***TCR*** V gene expression on CD8+ cytotoxic T lymphocytes from ***HIV*** -1-infected individuals. This was done using monoclonal ***antibodies*** against individual ***TCR*** V regions to trigger redirected cytolysis in 51Cr release assays. The percent specific lysis induced by each ***antibody*** functionally measures the representation of the ***TCR*** V region gene product it is specific for. Relative to non- ***HIV*** -infected controls and asymptomatic ***HIV*** -infected individuals with only moderate CD4 lymphocyte depletion, ***HIV*** -infected individuals with low CD4 lymphocyte counts exhibited skewed patterns of ***TCR*** V region representation. Therefore, the perturbation within the CD8+ cytotoxic T lymphocyte repertoire in ***HIV*** infection appears to be selective based on ***TCR*** V region usage, increasingly so as disease progresses. The ***TCR*** V genes affected varied between different ***HIV*** -infected individuals and skewing detected in functional assays was not always apparent by flow cytometric analysis. These results suggest that ***HIV*** infection causes generalized effects on the T-cell repertoire, which are reflected in the relative ***TCR*** V gene representation of the CD8+ cytotoxic T lymphocyte population in peripheral blood.

L4 ANSWER 13 OF 20 MEDLINE

95062162 Document Number: 95062162. PubMed ID: 7971973.

Autoantibodies to the alpha/beta T-cell receptors in ***human*** ***immunodeficiency*** ***virus*** infection: ***dysregulation*** and mimicry. Lake D F; Schluter S F; Wang E; Bernstein R M; Edmundson A B; Marchalonis J J. (College of Medicine, University of Arizona, Tucson 85724.) PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1994 Nov 8) 91 (23) 10849-53. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States. Language: English.

AB Autoimmune reactivity is a consequence of infection with ***human*** ***immunodeficiency*** ***virus*** (***HIV***). We studied serological cross-reactions of purified pooled IgG from sera of ***HIV*** -infected individuals by using nested sets of synthetic overlapping peptides duplicating the covalent structures of T-cell receptors (TCRs) and immunoglobulin light chains and report that two processes of ***autoantibody*** production occur. (i) IgG ***autoantibodies*** to putative regulatory variable domain CDR1 and FR3 epitopes (where CDR is complementarity-determining region and FR is framework region) are present in pooled IgG from ***HIV*** -infected individuals at levels 10-fold greater than that in pooled IgG from healthy humans. (ii) Anti- ***TCR*** autoimmunization involves antigenic mimicry between a conserved peptide stretch of the major neutralizing V3 loop determinant of ***HIV*** -1 gp120 and the conserved FR4 segment of the ***TCR*** V beta. Affinity-purified ***antibodies*** to the synthetic V3 loop peptide bound to a recombinant single-chain ***TCR*** and to a synthetic ***TCR*** joining segment peptide containing the FR4 sequence. Conversely, affinity-purified ***autoantibodies*** from pooled IgG from ***HIV*** -infected individuals to the ***TCR*** peptide bound the V3 loop peptide and a single-chain ***TCR*** . Inhibition studies indicated that the cross-reactive immunizing antigen was the V3 loop. These results bear upon the impact of ***HIV*** infection on immune regulation and on the selection of peptides for vaccine development.

L4 ANSWER 10 OF 20 MEDLINE

95285607 Document Number: 95285607. PubMed ID: 7768037. Autoreactivity in
HIV -1 infection: the role of molecular mimicry. Silvestris F;
Williams R C Jr; Dammacco F. (Department of Biomedical Sciences and Human
Oncology, University of Bari, Italy.) CLINICAL IMMUNOLOGY AND
IMMUNOPATHOLOGY, (1995 Jun) 75 (3) 197-205. Ref: 59. Journal code: DEA;
0356637. ISSN: 0090-1229. Pub. country: United States. Language: English.

AB Autoimmunity during ***HIV*** -1 infection may contribute to the
immunopathogenesis of AIDS. Titers of ***autoantibodies*** to
HLA molecules and other surface markers of CD4+ T cells appear to increase
with the progression of disease and may correlate with lymphopenia. Other
autoantibodies are directed at a number of regulatory molecules of
the immune system. Genesis of autoreactivity may be related to structural
homologies of ***HIV*** -1 env-products to such functional molecules
involved in the control of self-tolerance. The most impressive
similarities include the HLA-DR4 and DR2, the variable regions of
TCR alpha-, beta-, and gamma-chain, the Fas protein, and several
functional domains of IgG and IgA. Thus, ***HIV*** -1 infection may
induce ***dysregulation*** leading to autoimmune response, through a
number of molecular mimicry mechanisms. Pathogenicity of
antibodies to T cells could also include the activation of
membrane-to-nucleus signal transducers resulting in increased apoptosis.
The evolution of autoimmune mechanisms during ***HIV*** -1 infection
cannot exclude, however, progression to immunoproliferative malignancy,
since aspects of oligoclonal immune response to ***HIV*** -1 components
may occur in several autoimmune diseases which in some instances evolve to
lymphoma.

L4 ANSWER 1 OF 20 MEDLINE

2001495648 Document Number: 21429276. PubMed ID: 11544282. ***HIV***
-1 infection impairs cell cycle progression of CD4(+) T cells without
affecting early activation responses. Sieg S F; Harding C V; Lederman M M.
(Department of Medicine, Division of Infectious Diseases, Center for AIDS
Research, Case Western Reserve University, Cleveland, Ohio, USA.) JOURNAL
OF CLINICAL INVESTIGATION, (2001 Sep) 108 (5) 757-64. Journal code: HS7;
7802877. ISSN: 0021-9738. Pub. country: United States. Language: English.

AB Failure of CD4(+) T cells to proliferate in response to antigenic
stimulation is a characteristic of ***HIV*** infection. Analysis of
the proliferation defect has been hampered by an inability to identify
CD4(+) cells with ***T*** ***cell*** ***receptor***
specificity for antigen. To focus only on cells that had been stimulated
through the ***T*** ***cell*** ***receptor***, CD4(+) T cells
were stimulated with an anti-Vbeta3 Ab that activates approximately 3-5%
of peripheral blood T cells. This approach revealed proliferation defects
in cells from ***HIV*** -infected patients that were not appreciated
using anti-CD3 Ab stimulation and provided the capacity to examine
responses on a single cell basis. After anti-Vbeta3 Ab stimulation,
CD4(+)Vbeta3(+) cells from ***HIV*** -infected patients demonstrated
defects in expression of cell cycle-associated proteins, D-type cyclins,
and cyclin A. However, the expression of early activation markers, CD69
and CD25, was not significantly impaired in cells from most patients.
Thus, CD4(+) T cell proliferation failure in ***HIV*** disease is
characterized by dysregulated activation that precludes cell cycle
progression. This proliferation defect was most apparent in patients with
diminished CD4(+) T cell numbers and higher plasma ***HIV*** RNA
levels. CD4(+) T cell proliferation failure may be a key determinant of
immune ***impairment*** in ***HIV*** disease.

L6 ANSWER 7 OF 9 MEDLINE

95207724 Document Number: 95207724. PubMed ID: 7899820. The ***T***
cell ***receptor*** and AIDS pathogenesis. Hoffmann G W.
(Department of Microbiology and Immunology, University of British
Columbia, Vancouver, Canada.) SCANDINAVIAN JOURNAL OF IMMUNOLOGY, (1995
Apr) 41 (4) 331-7. Journal code: UCW; 0323767. ISSN: 0300-9475. Pub.
country: ENGLAND: United Kingdom. Language: English.

AB An idiotypic network model of AIDS pathogenesis is described in which the
T ***cell*** ***receptor*** plays a role both in infection
and as a target of autoimmunity. This is an extension of a previously
published autoimmunity model, and provides explanations for several
otherwise puzzling aspects of AIDS pathogenesis. In the model ***HIV***
-specific T cells are preferentially infected, and ***HIV***, acting
as an antigen, stimulates the expansion of the infectable pool of T cells.
The ***HIV*** variants that are most strongly selected are those that
are recognized by the most helper T cells. ***HIV*** and suppressor T
cells are subject to the same selective environment, and consequently
undergo a process of convergent selection to resemble each other more and
more with time. Eventually immunity against ***HIV*** cross-reacts
with suppressor T cell idiotypes, disrupting the normal regulation of
helper T cells. Autoimmunity ensues. The model leads to novel vaccine and
therapy approaches involving the targeting and elimination of ***HIV***
-specific T cells.

L6 ANSWER 2 OF 9 MEDLINE
97153482 Document Number: 97153482. PubMed ID: 9000486. Analysis of
autoantibodies to T-cell receptors among ***HIV*** -infected
individuals: epitope analysis and time course. Marchalonis J J; Ampel N M;
Schluter S F; Garza A; Lake D F; Galgiani J N; Landsperger W J. (College
of Medicine, University of Arizona, Tucson, Arizona, 85724, USA.)
CLINICAL IMMUNOLOGY AND IMMUNOPATHOLOGY, (1997 Feb) 82 (2) 174-89.
Journal code: DEA; 0356637. ISSN: 0090-1229. Pub. country: United States.
Language: English.

AB Individuals seropositive for ***human*** ***immunodeficiency***
virus type 1 (***HIV***) express elevated levels of
autoantibodies (AAbs) directed against recombinant T-cell
receptors (TCRs) and synthetic peptide epitopes duplicating beta chain
markers. We performed longitudinal studies of anti- ***TCR*** AAbs in
HIV -1-infected individuals, making comparisons with uninfected
sera and sera from other individuals infected with a nonviral agent. We
determined levels of ***autoantibodies*** by titration using
enzyme-linked immunosorbent assay (ELISA) and developed a means for
characterizing " ***autoantibody*** CDR recognition spectrotypes" for
individual sera. Antibody levels against certain defined synthetic
epitopes were substantially elevated in ***HIV*** -infected subjects
relative to reactivities by control groups. Individual sera showed
relatively high AAb levels to a subset of CDR1 peptide epitopes. Two
patients who subsequently developed AIDS showed particular reactivity to
Vbeta2.1, 8.1, 10.1, and 22.1 epitopes. Our results show that production
AAbs to ***TCR*** Vbeta epitopes is a general consequence of
HIV infection. The response is individual but shows some
restriction and shifts in AAb subpopulations often occur with time.

L15 ANSWER 2 OF 19 MEDLINE
2001197651 Document Number: 21151071. PubMed ID: 11256574. Emerging
principles for ***T*** ***cell*** ***receptor*** recognition
of antigen in cellular immunity. Garcia K C; Degano M; Speir J A; Wilson I
A. (The Scripps Research Institute, Department of Molecular Biology, La
Jolla, CA 92037, USA.) Rev Immunogenet, (1999) 1 (1) 75-90. Ref: 75.
Journal code: D09; 100883703. ISSN: 1398-1714. Pub. country: Sweden.

Language: English.

AB The structural basis of antigen recognition in cellular immunity has been elucidated through the determination of crystal structures of major histocompatibility complex (MHC) molecules bound to antigenic peptides, T cell receptors (***TCR***), CD8 and CD4 co-receptors and, most recently, TCRs in complex with peptide-MHC (pMHC). The mechanisms that generate the diversity of the immune response to invading microorganisms were first realized at a genetic level and are necessary in order to cope with the enormous number of potential antigens. This diversity is manifested in the protein products of the genes which code for the components of the ***TCR*** signalling complex. The structure of the ***TCR*** reveals both striking similarities with and fundamental differences from its ***functional*** counterpart, the antibody, in the humoral immune system. The conserved manner in which the ***TCR*** recognizes and interacts with its peptide-MHC ligand allows the ***TCR*** great latitude in its potential to form productive interactions with antigen-presenting cells that bear numerous ligands to which the ***TCR*** has not been previously exposed. This phenomenon of cross-, or alloreactivity arises from a combination of conserved structural features across all MHC molecules, both self and foreign, and some degree of molecular mimicry. Non-classical MHC ligands presenting either modified or specialized peptides, lipids, carbohydrates, or no ligand at all, are now thought to play increasingly important roles in cellular immunity. We ***review*** some of the recent structural results and our current state of knowledge about ***TCR*** structure, and how this relates to its ***function*** .

L15 ANSWER 10 OF 19 MEDLINE
97077676 Document Number: 97077676. PubMed ID: 8920243. Variant
TCR ligands: new insights into the molecular basis of antigen-dependent signal transduction and T-cell activation. Madrenas J; Germain R N. (Laboratory of Immunology, National Institute of Allergy and Infectious Diseases, Bethesda, MD 20892-1892, USA.) SEMINARS IN IMMUNOLOGY, (1996 Apr) 8 (2) 83-101. Ref: 99. Journal code: A61; 9009458. ISSN: 1044-5323. Pub. country: United States. Language: English.

AB Recent studies have identified peptide-MHC molecule ligands of alpha beta T-cell receptors with properties apparently distinct from classical agonists. These complexes, which are slight structural variants of the immunizing peptide or original presenting MHC molecule, have several novel properties. They can act as partial agonists able to induce only some and not other effector activities of the T cell, as antagonists able to inhibit T-cell ***functions*** stimulated by agonist ligand, or as mixed partial agonists/antagonists. Here we discuss the existing data suggesting that a simple receptor occupancy model does not account for the properties of these ***TCR*** ligands and ***review*** emerging data on qualitative differences in signal transduction following ***TCR*** engagement by priming versus variant complexes. We propose several non-exclusive models to explain both the biochemical and biological properties of variant ligands with partial agonist or antagonist properties.

L15 ANSWER 11 OF 19 MEDLINE
96303221 Document Number: 96303221. PubMed ID: 8732480. ***TCR*** gene polymorphisms and autoimmune disease. Kay R A. (Department of Pathology, Ninewells Hospital & Medical School, Dundee, UK.) EUROPEAN JOURNAL OF IMMUNOGENETICS, (1996 Apr) 23 (2) 161-77. Ref: 129. Journal code: AZ6; 9106962. ISSN: 0960-7420. Pub. country: ENGLAND: United Kingdom. Language: English.

AB Autoimmunity may result from abnormal regulation within the immune system. As the T cell is the principal regulator of the immune system and its normal ***function*** depends on immune recognition or self/non-self discrimination, abnormalities of the idiotypic ***T*** - ***cell*** ***receptor*** (***TCR***) may be one cause of autoimmune disease. The ***TCR*** is a clonally distributed, cell-surface heterodimer which binds peptide antigen when complexed with HLA molecules. In order to recognize the variety of antigens it may possibly encounter, the ***TCR***, by necessity, is a diverse structure. As with immunoglobulin, it is the variable domain of the ***TCR*** which interacts with antigen and exhibits the greatest amount of amino acid variability. The underlying genetic basis for this structural diversity is similar to that described for immunoglobulin, with ***TCR*** diversity relying on the somatic recombination, in a randomly imprecise manner, of smaller gene segments to form a ***functional*** gene. There are a large number of gene segments to choose from (particularly the TCRAV, TCRAJ and TCRBV gene segments) and some of these also exhibit allelic variation. Finally, polymorphisms in non-coding regions of ***TCR*** genes, leading to biased recombination or expression, are also beginning to be recognized. All these factors contribute to the polymorphic nature of the ***TCR***, in terms of both structure and repertoire formation. It follows that inherited abnormalities in either coding or regulatory regions of ***TCR*** genes may predispose to aberrant T-cell ***function*** and autoimmune disease. This ***review*** will outline the genomic organization of the ***TCR*** genes, the genetic mechanisms responsible for the generation of diversity, and the results of investigations into the association between germline polymorphisms and autoimmune disease.

L15 ANSWER 15 OF 19 MEDLINE
94137363 Document Number: 94137363. PubMed ID: 8305133. Tickling the ***TCR*** : selective T-cell ***functions*** stimulated by altered peptide ligands. Evavold B D; Sloan-Lancaster J; Allen P M. (Dept of Pathology, Washington University School of Medicine, St Louis, MO 63110.) IMMUNOLOGY TODAY, (1993 Dec) 14 (12) 602-9. Ref: 60. Journal code: AEA; 8008346. ISSN: 0167-5699. Pub. country: ENGLAND: United Kingdom. Language: English.

AB Recent observations of T-cell responses following ***T*** - ***cell*** ***receptor*** (***TCR***) interaction with altered peptide ligands have highlighted the complexity of this signalling system. The indications are that the ***TCR*** responds to minor changes in ligand with gradations of T-cell activation and effector ***functions***. Brian Evavold, Joanne Sloan-Lancaster and Paul Allen ***review*** these studies and present a model in which partial T-cell activation and ***TCR*** antagonism are related events in a continuum of signalling through the ***TCR***.

L15 ANSWER 17 OF 19 MEDLINE
92273160 Document Number: 92273160. PubMed ID: 1591001. The human ***T*** ***cell*** ***receptor*** in health and disease. Moss P A; Rosenberg W M; Bell J I. (Institute of Molecular Medicine, John Radcliffe Hospital, Headington, Oxford, United Kingdom.) ANNUAL REVIEW OF IMMUNOLOGY, (1992) 10 71-96. Ref: 139. Journal code: ALO; 8309206. ISSN: 0732-0582. Pub. country: United States. Language: English.

AB The T cell antigen receptor (***TCR***) recognizes antigen in the form of short peptides bound to a major histocompatibility (MHC) molecule. This ***review*** provides a synopsis of the current state of knowledge of the structure and ***function*** of the receptor and its possible role in human disease. Analysis of the ***T*** ***cell***

receptor usage of T-cell lines and clones recognizing the same peptide-MHC complex is beginning to shed light onto the structural basis of the ***TCR*** -peptide-MHC complex. Also, it is now apparent that there are two mechanisms by which the ***TCR*** can interact with the MHC molecule, either through classical peptide interactions or through super-antigens. Finally, we ***review*** the role of specific TCRs in human disease. Current evidence in this area is difficult to interpret; however, it is likely that ***TCR*** -mediated disease susceptibility exists, and its basis at either a germline or somatic level will soon be clarified.

L24 ANSWER 1 OF 3 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 1999-357172 [30] WPIDS
DNC C1999-105556
TI Delaying onset of AIDS in a host infected with an immunodeficiency-type
retrovirus.
DC B04
IN DEGHANPISHEH, K; HUANG, D S; ***MARCHALONIS, J J*** ; WANG, Y; WATSON,
R R
PA (ARIZ-N) ARIZONA BOARD OF REGENTS
CYC 1
PI US 5911990 A 19990615 (199930)* 13p
ADT US 5911990 A US 1996-696049 19960813
PRAI US 1996-696049 19960813

AB US 5911990 A UPAB: 19990802
NOVELTY - Administration of a peptide (I) corresponding to the first
complementarity determining region of T-cell receptor V beta (TCR V beta
CDR1) to a host infected with an immunodeficiency type retrovirus results
in the prevention of retrovirus-induced suppression of immune responses
and normalizes cytokine production.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for:
(1) a method of modulating the immune response of a mammal infected
with a C-type retrovirus or a lentivirus by administering (I) through a
systemic route to stimulate production of T helper 1 (Th1) cytokines;
interleukin 2 and interferon- gamma , and to suppress the production of T
helper 2 (Th2); interleukins 5, 6 and 10 and immunoglobulin G; and
(2) a method of altering the immune system response of a host
suffering from an infectious disease by artificially introducing (I) by
injection into the host's bloodstream or immune system so that the immune
system is artificially induced to stimulate production of Th1 cytokines or
suppress production of Th2 derived cytokines.
ACTIVITY - Anti-HIV.
MECHANISM OF ACTION - Immunomodulating agent.
(I) reduces the imbalance of Th1 and Th2 cytokine production and
contributes to the normalization of the immune response which retards
development of immune dysfunction.
Female C57Bl/6 5 week old mice were set up in 3 treatments: (1)
LP-BM5 retrovirus infected mice injected with saline, (2) LP-BM5
retrovirus infected mice injected with control peptide MCG3, or (3) LP-BM5
retrovirus infected mice injected with TCR V beta CDR1. Administration of
peptides (200 micro g/mice in saline, i.p.) was carried out twice on days
7 and 3 before retrovirus infection. Peptide preparations were free of
endotoxins and for TCR V beta CDR1 comprised the sequence Cys Lys Pro Ile
Ser Gly His Asn Ser Leu Phe Trp Tyr Arg Gln Thr corresponding to the human
TCR V beta sequence and for the control sequence Thr Gly Thr Ser Ser Asp
Val Gly Gly Tyr Asn Tyr Val Ser Trp Tyr corresponding to the CDR1 of the L
chain MCG. The LP-BM5 retrovirus was administered i.p. to mice on 0.1 ml
with an electrophic titer of 4.5 log10 PFU/ml which leads to rapid
induction of clinical symptoms with virtually no latent phase.
After 42 days of retroviral infection immunological analysis was
carried out. Specific titers of serum IgG to TCR- beta and MCG3 were
determined by enzyme linked immunosorbent assay (ELISA). IgG production by
LPS-stimulated splenocytes, an indication of early retrovirus infection
during the progression to murine AIDS, was significantly reduced (p less
than 0.05) by TCR V beta administration before infection but
administration of control peptide MCG3 showed no effect.
Treatment of retrovirally infected mice with (I) did not result in a
significant increase in mean Ab titers compared with either retrovirally
infected saline treated mice or retrovirally infected control treated
mice. In vitro production of Th1 cytokines, Il-2 and IFN- gamma by

concanavalin A-stimulated splenocytes was significantly (p less than 0.05) inhibited and normalized in the retrovirus infected mice administered with TCR V beta before infection but not in those administered the control peptide MCG3. Release of TNF- alpha by LPS-stimulated splenocytes was significantly inhibited in retrovirally infected mice administered with TCR V beta (p less than 0.05).

USE - Administration of (I) is used to delay the onset of AIDS through the restoration of normal levels of Th1 cytokines and Th2 derived cytokines and extends the period that occurs between infection by the retrovirus and the appearance of immune deficiencies. It causes the deleterious effects of infection to be reversed through prevention of immunosuppression and cytokine dysregulation that is otherwise induced by infection with an immunodeficiency-type retrovirus.

Studying (I) gives an insight into the pathogenesis occurring that leads to AIDS and into the idiotypic networks that involve autoantibodies and autoreactive T cells as regulatory elements and an understanding of the role of Vitamin E in immune function during retrovirus infection.

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OM protein - protein search, using sw model

Run on: February 9, 2002, 10:50:13 ; Search time 23.89 Seconds

(Without alignments)
49.610 Million cell updates/sec

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Gapop 10.0 , Gapext 0.5

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Total number of hits satisfying chosen parameters: 522463

Minimum DB seq length: 0
Maximum DB seq length: 200000000

Post-processing: Minimum Match 0%
Maximum Match 100%

Listing first 45 summaries

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Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

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3	96	100.0	312	7	AA160471
4	82	85.4	312	15	AA153145
5	80	83.3	114	13	AA126960
6	74	77.1	79	13	AA126969
7	74	77.1	92	13	AA163454
8	74	77.1	93	20	AA123521
9	74	77.1	94	16	AA178687
10	74	77.1	96	16	AA178687
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18	74	77.1 <td>314</td> <td>20</td> <td>AA199374</td> <td>Human T-cell recep</td>	314	20	AA199374	Human T-cell recep
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24	72	75.0 <td>314</td> <td>20</td> <td>AA15229</td> <td>Human receptor pro</td>	314	20	AA15229	Human receptor pro
25	70	72.9	79	13	AA126964	Human T lymphocyte
26	70	72.9	114	15	AA165457	T-cell receptor V-
27	70	72.9	114	15	AA123534	Amino acid sequenc
28	70	72.9	117	18	AA125939	T-cell receptor V-
29	70	72.9	287	21	AA156079	Mouse H2-Dd/flu nu
30	70	72.9	287	21	AA156081	HLA-A2/RII gag res
31	70	72.9	287	21	AA157855	TCR beta chain and
32	70	72.9	287	21	AA157857	TCR beta chain and
33	69	71.9	113	15	AA165456	T-cell receptor V-
34	69	71.9	114	20	AA123523	Amino acid sequenc
35	68	70.8	312	21	AA169988	Human receptor-ass
36	65	67.7	92	16	AA191171	TCR Vbeta5.3. Hom
37	63	65.6	345	21	AA169986	Human receptor-ass
38	62	64.6	112	21	AA183752	Canine T-cell rece
39	62	64.6	112	21	AA180657	Canine TCR V-beta
40	62	64.6	113	21	AA183753	Canine T-cell rece
41	62	64.6	113	21	AA180658	Canine TCR V-beta
42	62	64.6	114	21	AA180585	Canine T-cell rece
43	62	64.6	114	21	AA183754	Canine T-cell rece
44	62	64.6	114	21	AA180650	Canine TCR V-beta
45	62	64.6	114	21	AA180659	Canine TCR V-beta

ALIGNMENTS

RESULT 1
AA17984 standard; peptide: 16 AA.

ID	AA17984	standard; peptide: 16 AA.
XX	AA17984:	
AC	AA17984:	
XX		
DT	09-AUG-1999 (first entry)	
XX		
DE	Peptide derived from human T-cell receptor Vbeta gene product.	
XX		
KW	T-cell receptor; TCR; Immunodeficiency type retrovirus; Immune response;	
KW	cytokine; C-type retrovirus; lentivirus; T helper 1; Th1; Interleukin;	
KW	Interferon-gamma; Th2; Immunoglobulin G; AIDS; Immune deficiency;	
KM	Immunosuppression; cytokine dysregulation; TCR Vbeta gene.	
XX		
OS	Homo sapiens.	
XX		
PN	US911990-A.	
XX		
PD	15-JUN-1999.	
XX		
PF	13-AUG-1996; 96US-0696049.	
XX		
PR	13-AUG-1996; 96US-0696049.	
XX		
PA	(ARIZ-) ARIZONA BOARD OF REGENTS.	
XX		
PI	Dehghanipshch K, Huang DS, Marchalonis JJ, Wang Y;	
XX	Watson KR;	
XX		
DR	WPI: 1999-357172/30.	
XX		
PT	Delaying onset of AIDS in a host infected with an	
PT	Immunodeficiency-type retrovirus	

XX Claim 3; Column 9; 13pp; English.
PS
XX The invention relates to a peptide (I) corresponding to the first
CC complementarity determining region of T-cell receptor V beta (TCR V beta
CC Administering this peptide to a host infected with an immunodeficiency
CC type retrovirus results in the prevention of retrovirus-induced
CC suppression of immune responses and normalizes cytokine production. The
CC invention describes (1) a method of modulating the immune response of a
CC mammal infected with a C-type retrovirus or a lentivirus by administering
CC the TCR peptide through a systemic route to stimulate production of T
CC helper 1 (Th1) cytokines; interleukin 2 and interferon-gamma, and to
CC suppress the production of T helper 2 (Th2); interleukins 5, 6 and 10 and
CC immunoglobulin G; and (2) a method of altering the immune system response
CC of a host suffering from an infectious disease by artificially
CC introducing the peptide by injection into the host's bloodstream or
CC immune system so that the immune system is artificially induced to
CC stimulate production of Th1 cytokines or suppress production of Th2
CC derived cytokines. Administration of the peptide is used to delay the
CC onset of AIDS through the restoration of normal levels of Th1 cytokines
CC and Th2 derived cytokines and extends the period that occurs between
CC infection by the retrovirus and the appearance of immune deficiencies. It
CC causes the deleterious effects of infection to be reversed through
CC prevention of immunosuppression and cytokine dysregulation that is
CC otherwise induced by infection with an immunodeficiency-type retrovirus.
CC The present sequence represents the peptide of the invention derived from
CC TCR Vbeta gene product.
XX
SQ Sequence 16 AA:

Query Match 100.0%; Score 96; DB 20; Length 16;
Best Local Similarity 100.0%; Pred. No. 5.9e-09;
Matches 16; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGHSLEFWYROT 16
Db 1 CKPISGHSLEFWYROT 16

RESULT 2
AAP50079 standard; protein; 312 AA.
ID AAP50079 standard; protein; 312 AA.
XX
AC AAP50079;
XX
DT 19-MAR-1992 (first entry)
XX
XX T-cell antigen receptor protein.
XX
XX T-cell receptor; DNA probe; tumor marker; ss.
XX
XX EPI49548-A.
XX
XX 24-JUL-1985.
XX
XX 14-JAN-1985; 85EP-0300243.
XX
XX 06-FEB-1984; 84US-0577526.
XX
XX (ONTA-) ONTARIO CANCER INST.
XX
XX Mak TW;
XX
XX WPI: 1985-179193/30.
XX
XX N-PSDB: AAN50091.
XX
XX New nucleic acid encoding T-cell antigen receptor polypeptide -
XX useful for prep. of probes or antibodies for detection of
XX tumour cells and T-cells
XX
XX Disclosure: Fig 3; 15pp; English.
XX

CC This protein resembles human and mouse Ig light chain
CC molecules. This protein is encoded by clone Y135 and is
CC part of the antigen receptor mediating specialized T-
CC lymphocyte function. Antibodies may be directed against
CC this protein and used for detection of T-cell receptor
CC antigen so that unknown cells, e.g. a tumor cell, can be
CC identified as a T-cell or other cell.
XX
SQ Sequence 312 AA:

Query Match 100.0%; Score 96; DB 6; Length 312;
Best Local Similarity 100.0%; Pred. No. 1.3e-07;
Matches 16; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGHSLEFWYROT 16
Db 42 CKPISGHSLEFWYROT 57

RESULT 3
AAP60471 standard; protein; 312 AA.
ID AAP60471 standard; protein; 312 AA.
XX
AC AAP60471;
XX
DT 13-JUN-1991 (first entry)
XX
XX Portion of a human T-cell antigen receptor protein.
XX
XX Cancer; tumour cell; T-cell receptor.
XX
XX Homo sapiens.
XX
XX CA1197480-A.
XX
XX 03-DEC-1985.
XX
XX 01-FEB-1984; 84CA-0446545.
XX
XX 01-FEB-1984; 84CA-0446545.
XX
XX (ONTA-) ONTARIO CANCER INST.
XX
XX Mak TW;
XX
XX WPI: 1986-007147/02.
XX
XX N-PSDB: AAN60406.
XX
XX New pure nucleic acid with sequence encoding T-cell polypeptide -
XX is prep. by recombinant DNA methods for use as probe when
XX labelled and for antibody prodn.
XX
XX Claim 4; Fig 3; 22pp; English.
XX
XX Receptor protein product has at least 60% homology with the clone YT
XX 35. The product resembles human and mouse Ig light chain molecules,
XX and may be labelled for use as a probe for the detection of the
XX T-cell receptor antigen, and identification of unknown (esp. tumour
XX cells) as T-cells.
XX
SQ Sequence 312 AA:

Query Match 100.0%; Score 96; DB 7; Length 312;
Best Local Similarity 100.0%; Pred. No. 1.3e-07;
Matches 16; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGHSLEFWYROT 16
Db 42 CKPISGHSLEFWYROT 57

```

RESULT 4
ID AAR53145 standard; Protein: 312 AA.
XX
AC AAR53145:
DT 08-SEP-1994 (first entry)
XX
DE T-cell antigen receptor.
XX
KM T-cell antigen receptor; T-lymphocyte; probe; hybridization; MOLT-3.
XX
OS Homo sapiens.
XX
FH Key Location/Qualifiers
FT Misc-difference 90 /note= "possible N-glycosylation site"
FT Misc-difference 205 /note= "possible N-glycosylation site"
FT /note= "possible N-glycosylation site"
XX
N EP593092-A.
XX
PD 20-APR-1994.
XX
PF 14-JAN-1985; 85EP-0118895.
XX
PR 13-JAN-1984; 84US-0570694.
PR 06-FEB-1984; 84US-0577526.
XX
PA (ONTA-) ONTARIO CANCER INST.
XX
PI Mak TW;
XX
DR WP1; 1994-127936/16.
DR N-PSDB; AAQ62128.
XX
PT New nucleic acid encoding T-cell antigen receptor - is useful
PT e.g. as a probe to identify T-cells
XX
PS Disclosure; Fig 3; 13pp; English.
XX
CC mRNA complementary to the DNA sequence given in AAQ62128 is obtained by
CC isolating mRNA from MOLT-3 cells, preparing cDNA, inserting the cDNA
CC into the BglII site of vector pSP502EB5, transfecting the vector into
CC Escherichia coli HB101, and screening for 1.3 kb T-cell specific
CC mRNA in MOLT-3 and HSC-58 cells. The mRNA encodes a portion of the
CC T-cell antigen receptor (sequence AAR53145).
XX
SO Sequence 312 AA;

Query Match 85.4%; Score 82; DB 15; Length 312;
Best Local Similarity 93.8%; Pred. No. 2.3e-05;
Matches 15; Conservative 0; Mismatches 1; Indels 0; Gaps 0;

QY 1 CKPISGHSFLWYROT 16
   |||||
DB 42 ckpishnslfyrqt 57

RESULT 5
ID AAR26960 standard; Protein: 114 AA.
XX
AC AAR26960:
DT 11-FEB-1993 (first entry)
XX
DE Human T lymphocyte receptor V-beta w21 subfamily segment.
XX
KM TCR: IGR b 02; variable region; immunomodulation;
KM polymerase chain reaction; T cell receptor.
XX

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OS Homo sapiens.
XX
PN MO9213950-A.
XX
PD 20-AUG-1992.
XX
PF 12-FEB-1992; 92MO-FR00130.
XX
PR 12-FEB-1991; 91FR-0001613.
PR 12-APR-1991; 91FR-0004523.
XX
PA (ROUS ) ROUSSEL-UCLAF.
XX
PI Ferradini L, Hercend T, Roman-Roman S, Triebel F;
XX
DR WP1; 1992-300036/36.
DR N-PSDB; AAQ28173.
XX
PT Variable regions of b-chain of T-lymphocyte receptors and their
PT DNA - useful as immuno:modulant(s) and for diagnosing immune
PT disorders
XX
PS Claim 7; Page 37; 75pp; French.
XX
CC RNA was isolated from peripheral lymphocytes and converted to cDNA
CC using a C-beta-specific primer. The cDNA was amplified by anchored
CC PCR using C-beta and polyC primers, then amplified again using a
CC different C-beta specific primer. The amplified product was SacII-
CC restricted, inserted into Bluescript SK+ vector and used to transform
CC E.coli XL-1blue. Transformants were screened with a C-beta specific
CC probe and DNA from positive clones was sequenced in the C-beta
CC region. The sequence designated "IGR b 02" has ca. 85% homology
CC with the sequence HSTCRB23 (see Wilson R.K. et al., Immunogenetics
CC 32:406, 1990) and is a member of the Vbeta w21 subfamily. The
CC peptide encoded by it can be used to block T cell epitopes and in
CC vaccines. See also AAQ28174-028228.
XX
SO Sequence 114 AA;

Query Match 83.3%; Score 80; DB 13; Length 114;
Best Local Similarity 80.0%; Pred. No. 1.7e-05;
Matches 12; Conservative 2; Mismatches 1; Indels 0; Gaps 0;

QY 1 CKPISGHSFLWYRQ 15
   |||||
DB 42 ckpishnslfyrqt 56

RESULT 6
ID AAR26969 standard; Protein: 79 AA.
XX
AC AAR26969:
DT 11-FEB-1993 (first entry)
XX
DE Human T lymphocyte receptor V-beta 6 subfamily segment.
XX
KM TCR: IGR b 12; variable region; immunomodulation;
KM polymerase chain reaction; T cell receptor.
XX
OS Homo sapiens.
XX
PN MO9213950-A.
XX
PD 20-AUG-1992.
XX
PF 12-FEB-1992; 92MO-FR00130.
XX
PR 12-FEB-1991; 91FR-0001613.
PR 12-APR-1991; 91FR-0004523.
XX

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PA (ROUS) ROUSSEL-UCIAF.
 XX
 PI Ferradini L, Hercend T, Roman-Roman S, Triebel F;
 XX
 DR WPI: 1992-300036/36.
 DR N-PSDB: AAQ28182.
 XX
 PT Variable regions of b-chain of T-lymphocyte receptors and their
 PT DNA - useful as immuno:modulant(s) and for diagnosing immune
 PT disorders
 XX
 PS Claim 7; Page 46; 75pp; French.
 XX
 CC RNA was isolated from peripheral lymphocytes and converted to cDNA
 CC using a C-beta-specific primer. The cDNA was amplified by anchored
 CC PCR using C-beta and polyc primers, then amplified again using a
 CC different C-beta specific primer. The amplified product was SacII-
 CC restricted, inserted into Bluescript SK+ vector and used to transform
 CC E.coli XL-1blue. Transformants were screened with a C-beta specific
 CC probe and DNA from positive clones was sequenced in the C-beta
 CC region. The sequence designated "IGR b 12" represents a new member
 CC of the V beta 6 subfamily and has a homology of 94% with three
 CC previously identified members of the V beta 6 subfamily. The peptide
 CC encoded by it can be used to block T cell epitopes and in vaccines.
 CC See also AAQ28173-Q28228.
 CC
 CC
 CC Sequence 79 AA;
 XX
 SO
 QY 1 CKPISGHSLEFWYROT 16
 | | | | | : | : | | | |
 Db 7 cdplsghtalwytrgs 22
 77.1%; Score 74; DB 13; Length 79;
 Best Local Similarity 68.8%; Pred. No. 0.00011;
 Matches 11; Conservative 3; Mismatches 2; Indels 0; Gaps 0;

RESULT 7
 ID AAR65454
 AAR65454 standard; protein: 92 AA.
 XX
 AC AAR65454;
 XX
 DT 24-MAY-1995 (first entry)
 XX
 DE T-cell receptor V-beta HUVB6.9.
 XX
 V T-cell receptor; TCR; T-lymphocyte receptor; variable region;
 A beta-chain; V-beta; multiple sclerosis; cerebrospinal fluid;
 KM autoimmune disease; lymphoma; vaccine.
 XX
 OS Homo sapiens.
 PS
 XX WO9425063-A.
 PN
 XX 10-NOV-1994.
 PD
 XX 29-APR-1994; 94MO-US04789.
 PF
 XX 29-APR-1993; 93US-00055006.
 PR
 XX (IMMU-) IMMUNE RESPONSE CORP.
 PA (SAND-) SAN DIEGO REGIONAL CANCER CENT.
 XX
 PI Brostoff SM, Carlo DJ, Gold DP, Smith LR, Wilson DB;
 XX
 DR WPI: 1994-357913/44.
 DR
 XX New vaccine against multiple sclerosis using T-cell receptors
 PT or fragments of T-cell receptors from the beta chain variable
 PT region; for treating autoimmune disease and lymphoma(s)
 XX

PS Disclosure; Fig. 2A; 43pp; English.
 XX
 CC Sequences of the T-cell receptor beta-chain variable region that
 CC were most frequently expressed in cultures from the cerebrospinal
 CC fluid of multiple sclerosis patients are given in AAR65450-67. A
 CC peptide based on AAR65450-57 has been used for vaccine development.
 XX
 XX
 PS Sequence 92 AA;
 XX
 SO
 QY 1 CKPISGHSLEFWYROT 16
 | | | | | : | : | | | |
 Db 21 cdplsehmrlwytrgt 36
 77.1%; Score 74; DB 15; Length 92;
 Best Local Similarity 73.0%; Pred. No. 0.00012;
 Matches 12; Conservative 1; Mismatches 3; Indels 0; Gaps 0;

RESULT 8
 ID AAY23521
 AAY23521 standard; protein: 93 AA.
 XX
 AC AAY23521;
 XX
 DT 02-SEP-1999 (first entry)
 XX
 DE Amino acid sequence of human V beta 6.9.
 XX
 KW Vaccine; T cell receptor; TCR; T cell; V beta 6.2/3; V beta 6/5;
 KW V beta 6.7; V beta 2; V beta 5/1; V beta 7; V beta 13; V beta 6;
 KW multiple sclerosis.
 XX
 OS Homo sapiens.
 PS
 XX WO927957-A1.
 PN
 XX 10-JUN-1999.
 PD
 XX 03-DEC-1997; 97MO-US23147.
 PF
 XX 03-DEC-1997; 97MO-US23147.
 PR
 XX (IMMU-) IMMUNE RESPONSE CORP.
 PA (KIMM-) KIMMEL CANCER CENT SIDNEY.
 XX
 PI Brostoff SM, Carlo DJ, Gold DP, Smith LR, Wilson DB;
 XX
 DR WPI: 1999-404801/34.
 DR
 XX T0 cell receptor peptide-derived vaccines
 PT
 XX Example 8; Fig 2A-B; 104pp; English.
 PS
 XX The specification describes vaccines which comprise immunologically
 CC effective amounts of T cell receptor (TCR) peptides. The TCRs are
 CC present on the surface of T cells. The TCRs are chosen from V beta
 CC 6.2/3, V beta 6/5, V beta 6.7, V beta 2, V beta 5/1, V beta 7 or V beta
 CC 13. The V beta TCR peptide-based vaccines are useful for prevention or
 CC treatment of multiple sclerosis (MS). The presence of V beta 6.7 appears
 CC to be particularly associated with multiple sclerosis and can be used
 CC to determine an individual's susceptibility to multiple sclerosis.
 CC Vaccinating, rather than passively administering heterologous
 CC antibodies, allows the host's own immune system to mobilize and suppress
 CC auto aggressive T cells. Therefore, the suppression is persistent and
 CC may involve any and all immunological mechanisms in effecting that
 CC suppression. Such a multi-faceted response is more effective than
 CC the uni-dimensional suppression achieved by passive administration of
 CC monoclonal antibodies or extract-derived regulatory T cell clones.
 CC AAY23517-34 represent human V beta proteins that are most frequently
 CC expressed in the cerebrospinal fluid (CSF) of MS patients.
 XX
 SO Sequence 93 AA;

Query Match 77.1%; Score 74; DB 20; Length 93;
 Best Local Similarity 75.0%; Pred. No. 0.00013;
 Matches 12; Conservative 1; Mismatches 3; Indels 0; Gaps 0;

OY 1 CKPISGHNSLFMYROT 16
 1 1111111111111111
 DB 21 cdp1sehnrllywyrqt 36

RESULT 9

AAR78686
 ID AAR78686 standard; Protein: 94 AA.

AC AAR78686;

DT 12-APR-1996 (first entry)

XX T-cell receptor beta-chain variable region V-beta-6.1.

DE Diabetes; Immunotherapy; T-cell receptor beta-chain.

OS Homo sapiens.

XX Key Location/Qualifiers

FT Region 46..59
 FT /label= CDR2
 FT /note= "Claim 7, page 27"

PN WO9521623-A1.

PD 17-AUG-1995.

XX 10-FEB-1995; 95WO-US01572.

XX 14-FEB-1994; 94US-0195963.

XX (UYVE-) UNIV VERMONT.

PI Albertini RJ, Falta MT;

DR WPI: 1995-292941/38.

DR N-PSDB: AAO96132.

XX Preventing or reducing severity of diabetes - by inhibiting the

PT activity of specific T-cells, partic. by interfering with

PT diabetes-associated T cell receptors

XX Claim 4; Fig 1; 42pp; English.

XX Non-conserved regions of T-cell receptor (TCR) beta-chain variable

CC regions V-beta-6.1, V-beta-6.6/6.7 and V-beta-14 (AAR78686-88) are

CC used in the prevention or therapy of diabetes. Diabetes is

CC characterised by predominant usage of TCRs bearing V-beta-6 and

CC V-beta-14. Such TCRs can be administered to a patient to generate

CC an immune reaction that neutralises or kills V-beta-bearing T-cells.

CC Hypervariable CDR2 regions (see AAR75364, AAR78689-90) of the V-beta-6

CC and V-beta-14 chains may also be used.

XX Sequence 94 AA;

Query Match 77.1%; Score 74; DB 16; Length 94;

Best Local Similarity 68.8%; Pred. No. 0.00013;
 Matches 11; Conservative 3; Mismatches 2; Indels 0; Gaps 0;

OY 1 CKPISGHNSLFMYROT 16
 1 1111111111111111

DB 23 cdp1sghtalwytrqs 38

RESULT 10

AAR78687
 ID AAR78687 standard; Protein: 96 AA.

AC AAR78687;

DT 12-APR-1996 (first entry)

XX T-cell receptor beta-chain variable region V-beta-6.6./6.7.

DE Diabetes; Immunotherapy; T-cell receptor beta-chain.

OS Homo sapiens.

XX Key Location/Qualifiers

FT Region 48..61
 FT /label= CDR2
 FT /note= "Claim 8, page 28"

PN WO9521623-A1.

PD 17-AUG-1995.

XX 10-FEB-1995; 95WO-US01572.

XX 14-FEB-1994; 94US-0195963.

XX (UYVE-) UNIV VERMONT.

PI Albertini RJ, Falta MT;

DR WPI: 1995-292941/38.

DR N-PSDB: AAO96133.

XX Preventing or reducing severity of diabetes - by inhibiting the

PT activity of specific T-cells, partic. by interfering with

PT diabetes-associated T cell receptors

XX Claim 4; Fig 1; 42pp; English.

XX Non-conserved regions of T-cell receptor (TCR) beta-chain variable

CC regions V-beta-6.1, V-beta-6.6/6.7 and V-beta-14 (AAR78686-88) are

CC used in the prevention or therapy of diabetes. Diabetes is

CC characterised by predominant usage of TCRs bearing V-beta-6 and

CC V-beta-14. Such TCRs can be administered to a patient to generate

CC an immune reaction that neutralises or kills V-beta-bearing T-cells.

CC Hypervariable CDR2 regions (see AAR75364, AAR78689-90) of the V-beta-6

CC and V-beta-14 chains may also be used.

XX Sequence 96 AA;

Query Match 77.1%; Score 74; DB 16; Length 96;

Best Local Similarity 68.8%; Pred. No. 0.00013;
 Matches 11; Conservative 3; Mismatches 2; Indels 0; Gaps 0;

OY 1 CKPISGHNSLFMYROT 16
 1 1111111111111111

DB 24 cdp1sghtalwytrqs 39

RESULT 11

AAR65453
 ID AAR65453 standard; Protein: 104 AA.

AC AAR65453;

DT 24-MAY-1995 (first entry)

XX T-cell receptor V-beta HUVB6.5.

DE T-cell receptor; TCR; T-lymphocyte receptor; variable region;

KW beta-chain; V-beta; multiple sclerosis; cerebrospinal fluid;

KW autoimmune disease; lymphoma; vaccine.


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XX OS Homo sapiens.
XX PN W09425063-A.
XX PD 10-NOV-1994.
XX PE 29-APR-1994: 94WO-US04789.
XX PR 29-APR-1993: 93US-0055006.
XX PA (IMMU-) IMMUNE RESPONSE CORP.
XX PA (SAND-) SAN DIEGO REGIONAL CANCER CENT.
XX PI Brostoff SW, Carlo DJ, Gold DP, Smith LR, Wilson DB;
XX DR WPI: 1994-357913/44.
XX PT New vaccine against multiple sclerosis using T-cell receptors
XX or fragments of T-cell receptors from the beta chain variable
XX region; for treating auto-immune disease and lymphoma(s)
XX T
XX PS Disclosure: Fig. 2A: 43pp; English.
XX PS
XX CC Sequences of the T-cell receptor beta-chain variable region that
XX CC were most frequently expressed in cultures from the cerebrospinal
XX CC fluid of multiple sclerosis patients are given in AAR65450-67. A
XX CC peptide based on AAR65450-57 has been used for vaccine development.
XX CC
XX S0 Sequence 104 AA:

Query Match 77.18; Score 74; DB 15; Length 104;
Best Local Similarity 75.08; Pred. No. 0.00014;
Matches 12; Conservative 1; Mismatches 3; Indels 0; Gaps 0;

OY 1 CKPISGNSLFMYROT 16
   111111111111
Db 33 cdpisghnrlywyrqt 48

RESULT 12
AAY23520
ID AAY23520 standard; Protein: 106 AA.
XX AC AAY23520:
XX NT 02-SEP-1999 (first entry)
XX X
XX E Amino acid sequence of human V beta 6.5.
XX KW Vaccine; T cell receptor; TCR; T cell; V beta 6.2/3; V beta 6/5;
XX KW V beta 6.7; V beta 2; V beta 5/1; V beta 7; V beta 13; V beta 6;
XX KW multiple sclerosis.
XX OS Homo sapiens.
XX PN W09927957-A1.
XX PD 10-JUN-1999.
XX PE 03-DEC-1997: 97WO-US23147.
XX PR 03-DEC-1997: 97WO-US23147.
XX PA (IMMU-) IMMUNE RESPONSE CORP.
XX PA (KIMM-) KIMMEL CANCER CENT SIDNEY.
XX PI Brostoff SW, Carlo DJ, Gold DP, Smith LR, Wilson DB;
XX DR WPI: 1999-404801/34.
XX PT T0 cell receptor peptide-derived vaccines

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XX PS Example 8: Fig 2A-B: 104pp; English.
XX CC The specification describes vaccines which comprise immunologically
XX CC effective amounts of T cell receptor (TCR) peptides. The TCRs are
XX CC present on the surface of T cells. The TCRs are chosen from V beta
XX CC 6.2/3, V beta 6/5, V beta 6.7, V beta 2, V beta 5/1, V beta 7 or V beta
XX CC 13. The V beta TCR peptide-based vaccines are useful for prevention or
XX CC treatment of multiple sclerosis (MS). The presence of V beta 6.7 appears
XX CC to be particularly associated with multiple sclerosis and can be used
XX CC to determine an individual's susceptibility to multiple sclerosis.
XX CC Vaccinating, rather than passively administering heterologous
XX CC antibodies, allows the host's own immune system to mobilize and suppress
XX CC auto aggressive T cells. Therefore, the suppression is persistent and
XX CC may involve any and all immunological mechanisms in effecting that
XX CC suppression. Such a multi-faceted response is more effective than
XX CC the uni-dimensional suppression achieved by passive administration of
XX CC monoclonal antibodies or extent-derived regulatory T cell clones.
XX CC AAY23517-34 represent human V beta proteins that are most frequently
XX CC expressed in the cerebrospinal fluid (CSF) of MS patients.
XX S0 Sequence 106 AA:

Query Match 77.18; Score 74; DB 20; Length 106;
Best Local Similarity 75.08; Pred. No. 0.00014;
Matches 12; Conservative 1; Mismatches 3; Indels 0; Gaps 0;

OY 1 CKPISGNSLFMYROT 16
   111111111111
Db 34 cdpisghnrlywyrqt 49

RESULT 13
AAR65450
ID AAR65450 standard; protein: 113 AA.
XX AC AAR65450:
XX DT 24-MAY-1995 (first entry)
XX DE T-cell receptor V-beta HUVB6.1.
XX KW T-cell receptor; TCR; T-lymphocyte receptor; variable region;
XX KW beta-chain; V-beta; multiple sclerosis; cerebrospinal fluid;
XX KW auto-immune disease; lymphoma; vaccine.
XX OS Homo sapiens.
XX PN W09425063-A.
XX PD 10-NOV-1994.
XX PE 29-APR-1994: 94WO-US04789.
XX PR 29-APR-1993: 93US-0055006.
XX PA (IMMU-) IMMUNE RESPONSE CORP.
XX PA (SAND-) SAN DIEGO REGIONAL CANCER CENT.
XX PI Brostoff SW, Carlo DJ, Gold DP, Smith LR, Wilson DB;
XX DR WPI: 1994-357913/44.
XX PT New vaccine against multiple sclerosis using T-cell receptors
XX or fragments of T-cell receptors from the beta chain variable
XX PT region; for treating auto-immune disease and lymphoma(s)
XX PS Disclosure: Fig. 2A: 43pp; English.
XX CC Sequences of the T-cell receptor beta-chain variable region that
XX CC were most frequently expressed in cultures from the cerebrospinal
XX CC fluid of multiple sclerosis patients are given in AAR65450-67. A

```

CC peptide based on AAR65450-57 has been used for vaccine development.
XX
50 Sequence 113 AA;

Query Match	77.18;	Score 74;	DB 15;	Length 113;
Best Local Similarity	68.88;	Pred. NO. 0.00015;		
Matches 11: Conservative	3;	Mismatches 2;	Indels 0;	Gaps 0;

```

Oy 1 CKPISGHNSLFWYROT 16
    1 1 1 1 1 : 1 : 1 1 1 :
Db 42 cdpisghtalwyrgs 57

```

RESULT 14
AAR65455
ID AAR65455 standard; protein; 113 AA

AC	AAR65455;
YX	
YT	24-MAY-1995 (first entry)
YV	

XX T-cell receptor; TCR; T-lymphocyte receptor; variable region
KW beta-chain; V-beta; multiple sclerosis; cerebrospinal fluid;
KW

OS Homo sapiens.
XX
PN W09425063-A.

XX 29-APR-1994; 94WO-US04789.
XX
XX

PA (IMMU-) IMMUNE RESPONSE CORP.
PA (SAND-) SAN DIEGO REGIONAL CANCER CENT
XX

AA WPI; 1994-357913/44.
DR
XX
PT New vaccine against multiple sclerosis using T-cell rece

1X region; for creating auto-immune disease and lymphoma(s)
 2S
 3XX Disclosure; Fig. 2A; 43pp; English.

CC were most frequently expressed in cultures from the cerebrosplinal
CC fluid of multiple sclerosis patients are given in AAR65450-67. A
CC peptide based on AAR65450-57 has been used for vaccine development
XX

Query Match	77.18;	Score 74;	DB 15;	Length 113;
Best Local Similarity	68.88;	Pred. No.	0.00015;	

OY	1	CKPISCHNSLEWYROT	16
	1		:
42	cdpischta vwrras	57	
db			

RESULT	15
AY23517	
ID	AY23517 standard. protein. 114 AA

AC AAY23517;

XX 02-SEP-1999 (first entry)
DT
XX

Amino acid sequence of human V beta 6.1.

KW Vaccine; T cell receptor; TCR; T cell; V beta 6.2/3; V beta 6/5;
KW V beta 6.7; V beta 2; V beta 5/1; V beta 7; V beta 13; V beta 6;
KW multiple sclerosis.
...

OS	Homo sapiens.
XX	
PN	W09927957-A1.

XX	03-DEC-1997;	97WO-US23147.
PF		
XX		

XX (IMMU-) IMMUNE RESPONSE CORP.
PA (KIMM-) KIMMEL CANCER CENT SYDNEY
PA
RU

XX WPI; 1999-404801/34.
DR
XX

XX The specification describes vaccines

13. The ν beta TCR peptide-based vaccines are useful for prevention or treatment of multiple sclerosis (MS). The presence of ν beta 6.7 appears

[illegible]

CC suppression. Such a multi-faceted response is more effective than
CC the uni-dimensional suppression achieved by passive administration of
CC monoclonal antibodies or extant-derived regulatory T cell clones.
CC AAY23517-34 represent human V beta proteins that are most frequently

XX	
SO	Sequence 114 AA;

1 CKPTGHSNLSLEWYROT 16
Best Local Similarity 68.8%; Pred. No 0.000016;
Matches 11; Conservative 3; Mismatches 2; Indels

Db 42 cdpisghtalywyrqs 57

Search completed: February 9, 2002, 10:51:05
Job time: 52 sec

GenCore version 4.5
Copyright (c) 1993 - 2000 CompuGen Ltd.

OM protein - protein search, using sw model

Run on: February 9, 2002, 10:50:13 ; Search time 12.47 Seconds

(Without alignments)
28.874 Million cell updates/sec

Title: US-09-591-789-1

Sequence: 1 CKPISGHSLEFWYRQT 16

Scoring table: BLOSUM62
Gapop 10.0 , Gapext 0.5

Searched: 212252 seqs, 22503292 residues

Total number of hits satisfying chosen parameters: 212252

Minimum DB seq length: 0
Maximum DB seq length: 200000000

Post-processing: Minimum Match 0%
Maximum Match 100%

Listing first 45 summaries

Database : Issued Patents-AA:*
1: /cgn2_6/prodata/2/1aa/5A-COMB.pep:*
2: /cgn2_6/prodata/2/1aa/5B-COMB.pep:*
3: /cgn2_6/prodata/2/1aa/5A-COMB.pep:*
4: /cgn2_6/prodata/2/1aa/5B-COMB.pep:*
5: /cgn2_6/prodata/2/1aa/PCTUS-COMB.pep:*
6: /cgn2_6/prodata/2/1aa/backfiles1.pep:*

Pred. No. is the number of results predicted by chance to have a
score greater than or equal to the score of the result being printed,
and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	Query Match	Length	DB ID	Description
1	96	100.0	16	2	US-08-696-049-1
2	96	100.0	102	3	US-08-466-368-9
3	74	77.1	310	3	US-08-897-097-4
4	74	77.1	311	3	US-08-897-097-3
5	74	77.1	314	3	US-08-897-097-1
6	65	67.7	92	1	US-08-181-4928-27
7	65	67.7	92	5	PCT-US95-00408-27
8	56	58.3	82	1	US-08-476-405A-25
9	56	58.3	121	4	US-08-341-560B-4
10	56	58.3	121	5	PCT-US93-03895-4
11	56	58.3	250	4	US-08-341-560B-6
12	56	58.3	250	4	PCT-US93-03895-6
13	56	58.3	306	4	US-09-082-593-2
14	47	49.0	18	1	US-08-476-405A-1
15	47	49.0	94	3	US-08-297-395-10
16	47	49.0	106	2	US-08-652-558-1
17	47	49.0	113	2	US-08-466-860-7
18	47	49.0	113	3	US-08-472-040A-7
19	47	49.0	113	4	US-08-276-776-7
20	47	49.0	113	4	US-08-471-209-7
21	47	49.0	154	1	US-08-246-242-7
22	47	49.0	391	5	PCT-US95-15696-2
23	46	47.9	101	2	US-08-466-860-75
24	46	47.9	101	3	US-08-472-040A-75
25	46	47.9	101	4	US-08-276-776-75
26	46	47.9	101	4	US-08-471-209-75
27	46	47.9	113	2	US-08-466-860-9

28	46	47.9	113	3	US-08-472-040A-9	Sequence 9, Appl1
29	46	47.9	113	4	US-08-276-776-9	Sequence 9, Appl1
30	46	47.9	113	4	US-08-471-209-9	Sequence 9, Appl1
31	45	46.9	528	3	US-08-904-871-5	Sequence 5, Appl1
32	45	46.9	748	3	US-08-904-871-6	Sequence 6, Appl1
33	45	46.9	748	3	US-08-904-871-13	Sequence 13, Appl1
34	44	45.8	248	4	US-08-341-560B-8	Sequence 8, Appl1
35	44	45.8	248	5	PCT-US93-03895-8	Sequence 8, Appl1
36	44	45.8	293	6	5189147-3	Patent No. 5189147
37	43	44.8	107	5	PCT-US94-07659-8	Sequence 8, Appl1
38	43	44.8	1095	4	US-09-112-096-15	Sequence 15, Appl1
39	42	43.8	106	2	US-08-652-558-48	Sequence 48, Appl1
40	42	43.8	153	2	US-08-652-558-41	Sequence 41, Appl1
41	41	42.7	17	2	US-08-454-236-10	Sequence 10, Appl1
42	41	42.7	90	2	US-08-454-236-4	Sequence 4, Appl1
43	41	42.7	112	2	US-08-454-236-3	Sequence 3, Appl1
44	41	42.7	119	4	US-08-477-247-11	Sequence 11, Appl1
45	41	42.7	120	4	US-08-341-560B-2	Sequence 2, Appl1

ALIGNMENTS

RESULT 1
US-08-696-049-1
Sequence 1, Application US/08696049
Patent No. 5911990
GENERAL INFORMATION:
APPLICANT: Marchalonis, John J.
APPLICANT: Watson, Ronald R.
APPLICANT: Denhamisheh, Kelvan
APPLICANT: Wang, Yuejian
APPLICANT: Huang, Dennis S.
TITLE OF INVENTION: T-Cell Receptor Peptides and Methods for
TITLE OF INVENTION: Preventing the Progression to AIDS in an Animal Model
NUMBER OF SEQUENCES: 2
CORRESPONDENCE ADDRESS:
ADDRESSEE: Arnold, White & Durkee
STREET: P.O. Box 4433
CITY: Houston
STATE: Texas
COUNTRY: USA
ZIP: 77210
COMPUTER READABLE FORM:
MEDIUM TYPE: Floppy disk
COMPUTER: IBM PC compatible
OPERATING SYSTEM: PC-DOS/MS-DOS
SOFTWARE: Patent In Release #1.0, Version #1.30
CURRENT APPLICATION DATA:
APPLICATION NUMBER: US/08/696,049
FILING DATE: Concurrently Herewith
CLASSIFICATION: 514
ATTORNEY/AGENT INFORMATION:
NAME: Kammerer, Patricia A.
REGISTRATION NUMBER: 29,775
TELEPHONE: 713/787-1400
TELECOMMUNICATION INFORMATION:
REFERENCE/DOCKET NUMBER: ARIZ:002
INFORMATION FOR SEQ ID NO: 1:
SEQUENCE CHARACTERISTICS:
LENGTH: 16 amino acids
TYPE: amino acid
STRANDEDNESS:
TOPOLOGY: linear
MOLECULE TYPE: peptide
US-08-696-049-1

Query Match 100.0% Score 96; DB 2; Length 16;
Best Local Similarity 100.0%; Pred. No. 6.9e-10;
Matches 16; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGNSLFMYROT 16
DB 1 CKPISGNSLFMYROT 16

RESULT 2

US-08-466-368-9
Sequence 9, Application US/08466368
Patent No. 6093539
GENERAL INFORMATION:
APPLICANT: Maddon, Paul J.
APPLICANT: Littman, Dan R.
APPLICANT: Chess, Leonard
APPLICANT: Axel, Richard
APPLICANT: Weiss, Robin
APPLICANT: McDougall, J. S.
TITLE OF INVENTION: DNA ENCODING THE T CELL SURFACE PROTEIN
TITLE OF INVENTION: T4 AND USE OF FRAGMENTS OF T4 IN THE TREATMENT OF AIDS
NUMBER OF SEQUENCES: 21
CORRESPONDENCE ADDRESS:
ADDRESSEE: Cooper & Dunham LLP
STREET: 1185 Avenue of Americas
CITY: New York
STATE: New York
COUNTRY: USA
ZIP: 10036
COMPUTER READABLE FORM:
MEDIUM TYPE: Floppy disk
OPERATING SYSTEM: IBM PC Compatible
SOFTWARE: Patent Release #1.0, Version #1.30
CURRENT APPLICATION DATA:
APPLICATION NUMBER: US/08/466,368
FILING DATE: 06-JUN-1995
CLASSIFICATION: 435
ATTORNEY/AGENT INFORMATION:
NAME: White, John P.
REGISTRATION NUMBER: 28,678
REFERENCE/DOCKET NUMBER: 24577-E1-B/JPM/AKC
TELECOMMUNICATION INFORMATION:
TELEPHONE: 212-278-0400
TELEFAX: 212-391-0525
INFORMATION FOR SEQ ID NO: 9:
SEQUENCE CHARACTERISTICS:
LENGTH: 102 amino acids
TYPE: amino acid
STRANDEDNESS: single
TOPOLOGY: linear
MOLECULE TYPE: protein
HYPOTHETICAL: YES
ANTI-SENSE: YES
FEATURE:
NAME/KEY: Active-site
LOCATION: 1..102
US-08-466-368-9

Query Match 100.0% Score 96: DB 3: Length 102;
Best Local Similarity 100.0% Pred. No. 5.2e-09;
Matches 16: Conservative 0: Mismatches 0: Indels 0: Gaps 0:

OY 1 CKPISGNSLFMYROT 16
DB 23 CKPISGNSLFMYROT 38

RESULT 3

US-08-897-097-4
Sequence 4, Application US/08897097
Patent No. 6054292
GENERAL INFORMATION:
APPLICANT: Hillman, Jennifer L.
APPLICANT: Corley, Neil C.

TITLE OF INVENTION: T-CELL RECEPTOR BETA-LIKE PROTEIN
NUMBER OF SEQUENCES: 4
CORRESPONDENCE ADDRESS:
ADDRESSEE: Incyte Pharmaceuticals, Inc.
STREET: 3174 Porter Drive
CITY: Palo Alto
STATE: CA
COUNTRY: USA
ZIP: 94304
COMPUTER READABLE FORM:
MEDIUM TYPE: Diskette
COMPUTER: IBM Compatible
OPERATING SYSTEM: DOS
SOFTWARE: FASTSEQ for Windows Version 2.0
CURRENT APPLICATION DATA:
APPLICATION NUMBER: US/08/897,097
FILING DATE: Herewith
CLASSIFICATION: 536
PRIOR APPLICATION DATA:
APPLICATION NUMBER:
FILING DATE:
ATTORNEY/AGENT INFORMATION:
NAME: Billings, Lucy J.
REGISTRATION NUMBER: 36,749
REFERENCE/DOCKET NUMBER: PF-0346 US
TELECOMMUNICATION INFORMATION:
TELEPHONE: 415-855-0555
TELEFAX: 415-845-4166
TELEX:
INFORMATION FOR SEQ ID NO: 4:
SEQUENCE CHARACTERISTICS:
LENGTH: 310 amino acids
TYPE: amino acid
STRANDEDNESS: single
TOPOLOGY: linear
IMMEDIATE SOURCE:
LIBRARY: GenBank
CLONE: 339012
US-08-897-097-4

Query Match 77.1% Score 74: DB 3: Length 310;
Best Local Similarity 75.0% Pred. No. 8e-05;
Matches 12: Conservative 1: Mismatches 3: Indels 0: Gaps 0:

OY 1 CKPISGNSLFMYROT 16
DB 42 CDPISGNSLFMYROT 57

US-08-897-097-3
Sequence 3, Application US/08897097
Patent No. 6054292
GENERAL INFORMATION:
APPLICANT: Hillman, Jennifer L.
APPLICANT: Corley, Neil C.
TITLE OF INVENTION: T-CELL RECEPTOR BETA-LIKE PROTEIN
NUMBER OF SEQUENCES: 4
CORRESPONDENCE ADDRESS:
ADDRESSEE: Incyte Pharmaceuticals, Inc.
STREET: 3174 Porter Drive
CITY: Palo Alto
STATE: CA
COUNTRY: USA
ZIP: 94304
COMPUTER READABLE FORM:
MEDIUM TYPE: Diskette
COMPUTER: IBM Compatible
OPERATING SYSTEM: DOS
SOFTWARE: FASTSEQ for Windows Version 2.0
CURRENT APPLICATION DATA:
APPLICATION NUMBER: US/08/897,097

US-08-897-097-4
Sequence 4, Application US/08897097
Patent No. 6054292
GENERAL INFORMATION:
APPLICANT: Hillman, Jennifer L.
APPLICANT: Corley, Neil C.
TITLE OF INVENTION: T-CELL RECEPTOR BETA-LIKE PROTEIN
NUMBER OF SEQUENCES: 4
CORRESPONDENCE ADDRESS:
ADDRESSEE: Incyte Pharmaceuticals, Inc.
STREET: 3174 Porter Drive
CITY: Palo Alto
STATE: CA
COUNTRY: USA
ZIP: 94304
COMPUTER READABLE FORM:
MEDIUM TYPE: Diskette
COMPUTER: IBM Compatible
OPERATING SYSTEM: DOS
SOFTWARE: FASTSEQ for Windows Version 2.0
CURRENT APPLICATION DATA:
APPLICATION NUMBER: US/08/897,097

```

1      FILING DATE: Herewith
2      CLASSIFICATION: 536
3      PRIOR APPLICATION DATA:
4      APPLICATION NUMBER:
5      FILING DATE:
6      ATTORNEY/AGENT INFORMATION:
7      NAME: Billings, Lucy J
8      REGISTRATON NUMBER: 36,749
9      REFERENCE/DOCKET NUMBER: PF-0346 US
10     TELECOMMUNICATION INFORMATION:
11     TELEPHONE: 415-855-0555
12     TELEFAX: 415-845-4166
13     TELEX:
14     INFORMATION FOR SEQ ID NO: 3:
15     SEQUENCE CHARACTERISTICS:
16     LENGTH: 311 amino acids
17     TYPE: amino acid
18     STRANDEDNESS: single
19     TOPOLOGY: linear
20     IMMEDIATE SOURCE:
21     LIBRARY: Genbank
22     CLONE: 1100182
23     US-08-897-097--3

```

```

Query Match 77.1%; Score 74; DB 3; Length 311;
Best Local Similarity 75.0%; Pred. No. 8,1e-05;
Matches 12; Conservative 1; Mismatches 3; Indels 0; Gaps 0;
OY 1 CKPIGSHNSLEFWYROT 16
    | | | | | | | | | |
DB 42 CDPISEHNRLWYVYROT 57
    | | | | | | | | | |

```

```

1      5
2  US-08-897-097-1
3  ; Sequence 1, Application US/08897097
4  ; Patent No. 6054292
5  ; GENERAL INFORMATION:
6  ; APPLICANT: Hillman, Jennifer L.
7  ; APPLICANT: Corley, Nell C.
8  ; TITLE OF INVENTION: T-CELL RECEPTOR BETA-LIKE PROTEIN
9  ; NUMBER OF SEQUENCES: 4
10 ; CORRESPONDENCE ADDRESS:
11 ; ADDRESSEE: Incyte Pharmaceuticals, Inc.
12 ; STREET: 3174 Porter Drive
13 ; CITY: Palo Alto
14 ; STATE: CA
15 ; COUNTRY: USA
16 ; ZIP: 94304
17 ; COMPUTER READABLE FORM:
18 ; MEDIUM TYPE: Diskette
19 ; COMPUTER: IBM Compatible
20 ; OPERATING SYSTEM: DOS
21 ; SOFTWARE: FASTEST for Windows Version 2.0
22 ; CURRENT APPLICATION DATA:
23 ; APPLICATION NUMBER: US/08/897,097
24 ; FILING DATE: Herewith
25 ; CLASSIFICATION: 536
26 ; PRIOR APPLICATION DATA:
27 ; APPLICATION NUMBER:
28 ; FILING DATE:
29 ; ATTORNEY/AGENT INFORMATION:
30 ; NAME: Billings, Lucy J.
31 ; REGISTRATION NUMBER: 36,749
32 ; REFERENCE/DOCKET NUMBER: PP-0346 US
33 ; TELECOMMUNICATION INFORMATION:
34 ; TELEPHONE: 415-845-0555
35 ; TELEFAX: 415-845-4166
36 ; TELEX:
37 ; INFORMATION FOR SEQ ID NO: 1:
38 ; SEQUENCE CHARACTERISTICS:
39 ; LENGTH: 314 amino acids

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:      TYPE: amino acid
:      STRANDEDNESS: single
:      TOPOLOGY: linear
:      IMMEDIATE SOURCE:
:      LIBRARY: TONGTUT01
:      CLONE: 983910
:
US-08-897-097-1

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Query Match	77.1%	Score 74:	DB 3:	Length 314:
Best Local Similarity	68.8%	Pred No	8.1e-05:	
Matches 11:	Conservative 3:	Mismatches 2:	Indels 0:	Gaps 0:

```
Qy      1 CKPISGHNLSFWYRQT 16
         | | | | | : | | | | :
Db      42 CDPISGHTALYWRQS 57
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RESULT 6
US-08-181-492B-27
; Sequence 27, Application US/08181492B
; Patent No. 5552300
; GENERAL INFORMATION:

TITLE OF INVENTION: T Cell Antigen Receptor V Region
 NUMBER OF SEQUENCES: 27
 CORRESPONDENCE ADDRESS:
 ADDRESSEE: T Cell Sciences, Inc.
 STREET: 115 Fourth Avenue
 CITY: Needham
 STATE: Massachusetts
 COUNTRY: United States of America
 ZIP: 02194-2725

```

1  MEDIM TYPE: Diskette, 3.50 Inch, 1.44mb storage
2  COMPUTER: IBM PC Compatible
3  OPERATING SYSTEM: MS-DOS 6.2
4  SOFTWARE: WordPerfect 6.1
5  CURRENT APPLICATION DATA:
6  APPLICATION NUMBER: US/08/181.492B
7  FILING DATE: 13-January-1994
8  CLASSIFICATION:
9  PRIOR APPLICATION DATA:
10 APPLICATION NUMBER:
11 FILING DATE:
12 ATTORNEY/AGENT INFORMATION:
13 NAME: Yankwich, Leon R.
14 REGISTRATION NUMBER: 30,237
15 REFERENCE/DOCKET NUMBER: TCS-203-P
16 TELECOMMUNICATION INFORMATION:
17 TELEPHONE: 617-345-9100
18 TELEFAX: 617-345-9111
19 INFORMATION FOR SEQ ID NO: 27:
20 SEQUENCE CHARACTERISTICS:
21 LENGTH: 92 amino acids
22 TYPE: amino acid
23 STRANDEDNESS: single
24 TOPOLOGY: linear
25 MOLECULE TYPE: protein
26 HYPOTHETICAL:
27 ANTI-SENSE:
28 FEATURE:
29 NAME/KEY:
30 LOCATION:
31 US-08-181-492B-27

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Query Match	67.7%	Score 65;	DB 1;	Length 92;
Best Local Similarity	66.7%;	Pred. No. 0.00067;		
Matches 10;	Conservative	2;	Mismatches 3;	Indels 0;
Caps	0;			
QY	1	CKPIGSHNSLPWYRQ	15	

COMPUTER READABLE FORM:
MEDIUM TYPE: FLOPPY disk
COMPUTER: IBM PC compatible
OPERATING SYSTEM: PC-DOS/MS-DOS
SOFTWARE: Patentin Release #1.0, Version #1.30
CURRENT APPLICATION DATA:
APPLICATION NUMBER: US/08/341,560B
FILING DATE: 17-Nov-1994
CLASSIFICATION: 435
PRIOR APPLICATION DATA:
APPLICATION NUMBER: US 07/963,333
FILING DATE: 19-OCT-1992
PRIOR APPLICATION DATA:
APPLICATION NUMBER: US 07/873,930
FILING DATE: 24-APR-1992
ATTORNEY/AGENT INFORMATION:
NAME: Parker, David L.
REGISTRATION NUMBER: 32,165
REFERENCE/DOCKET NUMBER: UTSD:353
TELECOMMUNICATION INFORMATION:
TELEPHONE: (512) 418-3000
TELEFAX: (713) 789-2679
INFORMATION FOR SEQ ID NO: 4:
SEQUENCE CHARACTERISTICS:
LENGTH: 121 amino acids
TYPE: amino acid
STRANDEDNESS:
TOPOLOGY: linear
US-08-341-560B-4

Query Match 58.3%; Score 56; DB 4; Length 121;
Best Local Similarity 46.7%; Pred. No. 0.029;
Matches 7; Conservative 4; Mismatches 4; Indels 0; Gaps 0;

Y 1 CKPISGNSLFWYRQ 15
| : ||::|||
Db 23 CNOTNNNNMYWYRQ 37

RESULT 10
PCT-US93-03895-4
Sequence 4, Application PC/TUS9303895
GENERAL INFORMATION:
APPLICANT: BOARD OF REGENTS, THE UNIVERSITY
OF TEXAS SYSTEM
APPLICANT: WARD, Elizabeth Sally and KIM, Jin-Kyoo
TITLE OF INVENTION: RECOMBINANT PRODUCTION OF
IMMUNOGLOBULIN-LIKE DOMAINS IN
PROKARYOTIC CELLS
NUMBER OF SEQUENCES: 16
CORRESPONDENCE ADDRESS:
ADDRESSEE: ARNOLD, WHITE & DURKEE
STREET: P.O. BOX 4433
CITY: HOUSTON
STATE: TEXAS
COUNTRY: USA
ZIP: 77210
COMPUTER READABLE FORM:
MEDIUM TYPE: FLOPPY disk
COMPUTER: IBM PC compatible
OPERATING SYSTEM: PC-DOS/MS-DOS
SOFTWARE: WORDPERFECT 5.1
CURRENT APPLICATION DATA:
APPLICATION NUMBER: PCT/US93/03895
FILING DATE: 19930426
CLASSIFICATION:
ATTORNEY/AGENT INFORMATION:
NAME: KITCHELL, BARBARA S.
REGISTRATION NUMBER: 33,928
REFERENCE/DOCKET NUMBER: UTPD353PCT
TELECOMMUNICATION INFORMATION:
TELEPHONE: 512-320-7200

TELEFAX: 512-474-7577
TELEX: 79-0924
INFORMATION FOR SEQ ID NO: 4:
SEQUENCE CHARACTERISTICS:
LENGTH: 121 amino acid residues
TYPE: AMINO ACID
STRANDEDNESS: single
TOPOLOGY: linear
PCT-US93-03895-4

Query Match 58.3%; Score 56; DB 5; Length 121;
Best Local Similarity 46.7%; Pred. No. 0.029;
Matches 7; Conservative 4; Mismatches 4; Indels 0; Gaps 0;

Y 1 CKPISGNSLFWYRQ 15
| : ||::|||
Db 23 CNOTNNNNMYWYRQ 37

RESULT 11
US-08-341-560B-6
Sequence 6, Application US/08341560B
Patent No. 6165745
GENERAL INFORMATION:
APPLICANT: Ward, E. Sally
APPLICANT: Kim, Jin-Kyoo
TITLE OF INVENTION: Recombinant Production of
Immunoglobulin-Like Domains in Prokaryotic Cells
NUMBER OF SEQUENCES: 33
CORRESPONDENCE ADDRESS:
ADDRESSEE: ARNOLD, WHITE & DURKEE
STREET: P.O. Box 4433
CITY: Houston
STATE: Texas
COUNTRY: USA
ZIP: 7721-4433
COMPUTER READABLE FORM:
MEDIUM TYPE: FLOPPY disk
COMPUTER: IBM PC compatible
OPERATING SYSTEM: PC-DOS/MS-DOS
SOFTWARE: Patentin Release #1.0, Version #1.30
CURRENT APPLICATION DATA:
APPLICATION NUMBER: US/08/341,560B
FILING DATE: 17-NOV-1994
CLASSIFICATION: 435
PRIOR APPLICATION DATA:
APPLICATION NUMBER: US 07/963,333
FILING DATE: 19-OCT-1992
PRIOR APPLICATION DATA:
APPLICATION NUMBER: US 07/873,930
FILING DATE: 24-APR-1992
ATTORNEY/AGENT INFORMATION:
NAME: Parker, David L.
REGISTRATION NUMBER: 32,165
REFERENCE/DOCKET NUMBER: UTSD:353
TELECOMMUNICATION INFORMATION:
TELEPHONE: (512) 418-3000
TELEFAX: (713) 789-2679
INFORMATION FOR SEQ ID NO: 6:
SEQUENCE CHARACTERISTICS:
LENGTH: 250 amino acids
TYPE: amino acid
STRANDEDNESS:
TOPOLOGY: linear
US-08-341-560B-6

Query Match 58.3%; Score 56; DB 4; Length 250;
Best Local Similarity 46.7%; Pred. No. 0.063;
Matches 7; Conservative 4; Mismatches 4; Indels 0; Gaps 0;

Y 1 CKPISGNSLFWYRQ 15

Db 152 CNOTNNNNMYWYRQ 166

RESULT 12

PCT-US93-03895-6

Sequence 6, Application PC/TUS9303895

GENERAL INFORMATION:

APPLICANT: BOARD OF REGENTS, THE UNIVERSITY

APPLICANT: OF TEXAS SYSTEM

APPLICANT: MARD, Elizabeth Sally and KIM, Jin-Kyoo

TITLE OF INVENTION: RECOMBINANT PRODUCTION OF

TITLE OF INVENTION: IMMUNOGLOBULIN-LIKE DOMAINS IN

TITLE OF INVENTION: PROKARYOTIC CELLS

NUMBER OF SEQUENCES: 16

CORRESPONDENCE ADDRESS:

ADDRESSEE: ARNOLD, WHITE & DURKEE

STREET: P.O. BOX 4433

CITY: HOUSTON

STATE: TEXAS

COUNTRY: USA

ZIP: 77210

COMPUTER READABLE FORM:

MEDIUM TYPE: FLOPPY DISK

OPERATING SYSTEM: PC-DOS/MS-DOS

SOFTWARE: WORDPERFECT 5.1

CURRENT APPLICATION DATA:

APPLICATION NUMBER: PCT/US93/03895

FILING DATE: 19930426

CLASSIFICATION:

ATTORNEY/AGENT INFORMATION:

NAME: KITCHELL, BARBARA S.

REGISTRATION NUMBER: 33,928

REFERENCE/DOCKET NUMBER: UTE9353PCT

TELECOMMUNICATION INFORMATION:

TELEPHONE: 512-320-7200

TELEFAX: 512-474-7577

INFORMATION FOR SEQ ID NO: 6:

SEQUENCE CHARACTERISTICS:

LENGTH: 250 amino acid residues

TYPE: AMINO ACID

STRANDEDNESS: single

TOPOLOGY: linear

PCT-US93-03895-6

Query Match

Best Local Similarity 46.7%;

Matches 7; Conservative 4;

Mismatches 4; Indels 0;

Caps 0;

QY 1 CKPISCHNSLFYWRQ 15

Db 152 CNOTNNNNMYWYRQ 166

RESULT 13

US-09-082-593-2

Sequence 2, Application US/09082593

Patent No. 6180104

GENERAL INFORMATION:

APPLICANT: DAVIS, MARK M.

APPLICANT: HEDRICK, STEPHEN M.

TITLE OF INVENTION: T CELL RECEPTOR BETA SUBUNIT

FILE REFERENCE: JX1193-195DIY2

CURRENT APPLICATION NUMBER: US/09/082,593

NUMBER OF SEQ ID NOS: 15

SOFTWARE: PatentIn Ver. 2.0

SEQ ID NO 2

LENGTH: 306

TYPE: PRT

ORGANISM: Mus musculus

RESULT 14

US-08-476-405A-1

Sequence 1, Application US/08476405A

Patent No. 5776459

GENERAL INFORMATION:

APPLICANT: Vandenbark, Arthur A.

APPLICANT: Method of Treatment Using TCR VBetas Peptides

TITLE OF INVENTION: Method of Treatment Using TCR VBetas Peptides

NUMBER OF SEQUENCES: 27

CORRESPONDENCE ADDRESS:

ADDRESSEE: Connective Therapeutics, Inc.

STREET: 3400 West Baysshore Road

CITY: Palo Alto

STATE: California

COUNTRY: USA

ZIP: 94303

COMPUTER READABLE FORM:

MEDIUM TYPE: FLOPPY disk

OPERATING SYSTEM: PC-DOS/MS-DOS

SOFTWARE: PatentIn Release #1.0, Version #1.25

CURRENT APPLICATION DATA:

APPLICATION NUMBER: US/08/476,405A

FILING DATE:

CLASSIFICATION: 424

PRIOR APPLICATION DATA:

APPLICATION NUMBER: US 08/059,020

FILING DATE: 16-MAR-1993

PRIOR APPLICATION DATA:

APPLICATION NUMBER: US 07/735,612

FILING DATE: 16-JUL-1991

PRIOR APPLICATION DATA:

APPLICATION NUMBER: US 07/708,022

FILING DATE: 31-MAY-1991

PRIOR APPLICATION DATA:

APPLICATION NUMBER: US 07/554,529

FILING DATE: 19-JUL-1990

PRIOR APPLICATION DATA:

APPLICATION NUMBER: US 07/467,577

FILING DATE: 19-JAN-1990

PRIOR APPLICATION DATA:

APPLICATION NUMBER: US 07/382,804

FILING DATE: 19-JUL-1989

PRIOR APPLICATION DATA:

ATTORNEY/AGENT INFORMATION:

NAME: Lowin, David A.

REGISTRATION NUMBER: 29,326

REFERENCE/DOCKET NUMBER: 886 P15

TELECOMMUNICATION INFORMATION:

TELEPHONE: 415-843-2800

TELEFAX: 415-843-2899

INFORMATION FOR SEQ ID NO: 1:

SEQUENCE CHARACTERISTICS:

LENGTH: 18 amino acids

TYPE: amino acid

STRANDEDNESS: single

TOPOLOGY: linear

MOLECULE TYPE: peptide

US-08-476-405A-1

Query Match

Best Local Similarity 43.8%;

Matches 7; Conservative 6;

Mismatches 3; Indels 0;

Caps 0;

QY 41 CEQHLGHNNAMWYWKOS 56

Db 41 CEQHLGHNNAMWYWKOS 56

Query Match 49.0%; Score 47; DB 1; Length 18;

Best Local Similarity 53.8%; Pred. No. 0.11;
Matches 7; Conservative 4; Mismatches 2; Indels 0; Gaps 0;

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Matches 7; Conservative 4; Mismatches 2; Indels 0; Gaps 0;
```

QY 3 PISCHNSLEWYRQ 15

```

Db      1 PKSGHDTVSWYQQ 13

```

RESULT 15
HE-09-207-

US-08-297-395-10

; Sequence 10, Application US/08297395A

; Patent NO. 6039947

; GENERAL INFORMATION:

APPLICANT: Howard L. Welner

APPLICANT: David A. Hafler

1. TITLE OF INVENTION: PEPTIDES DERIVED FROM IMMUNODOMINANT

1. TITLE OF INVENTION: EPITOPES OF MYELIN BASIC PROTEIN

FILE REFERENCE: 1010/05723US3

CURRENT APPLICATION NUMBER: US/

CURRENT FILING DATE: 1994-08-11

EARLIER APPLICATION NUMBER: 08/

EARLIER FILING DATE: 1993-05-06

EARLIER APPLICATION NUMBER: 07/

EARLIER FILING DATE: 1990-03-30

EARLIER APPLICATION NUMBER: PCT/US95/000000

EARLIER FILING DATE: 1988-06-24

EARLIER APPLICATION NUMBER: 07/
EARLIER FILING DATE: 1997-06-24

EARLIER FILING DATE: 1988

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; NUMBER OF SEO ID NOS: 84
SOFTWARE: FastSEO for Windows Version 3.0

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; SOFTWARE: F
; SEQ ID NO 10: SEQ ID NO 10
: LENGTH: 94

LENGTH: 94
TYPE: PRT

ORGANISM: Homo sapiens

US-08-297-395-10

Query Match 49.08; Score 47; DB 3; Length 94;

Best Local Similarity 40.08; Pred. No. 0.68;

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Matches 6; Conservative 5; Mismatches 4; Indels 0; Gaps 0;
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QY 1 CKPISCHNSLFWYRQ 15

— :: — :: — :: — :: — :: — ::

Search completed: February 9, 2002, 10:51:25
Job time: 72 sec

Job time: 72 sec

GenCore version 4.5
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OM protein - protein search, using sw model

Run on: February 9, 2002, 10:50:13 ; Search time 12.99 Seconds

(without alignments)
93.825 Million cell updates/sec

Title: US-09-591-789-1

Perfect score: 96

Sequence: 1 CKPISGHNSLFWYRQT 16

Scoring table: BLOSUM62

Gapop 10.0 , Gapext 0.5

Searched: 219241 seqs, 76174552 residues

Total number of hits satisfying chosen parameters: 219241

Minimum DB seq length: 0

Maximum DB seq length: 200000000

Post-processing: Minimum Match 0%

Maximum Match 100%

Listing first 45 summaries

Database :

1: PIR1.*
2: PIR2.*
3: PIR3.*
4: PIR4.*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	Query Match	Length	ID	Description
1	96	100.0	114	2	T-cell receptor be
2	96	100.0	115	2	T-cell receptor be
3	96	100.0	135	1	T-cell receptor be
4	96	100.0	135	2	T-cell receptor be
5	87	90.6	134	2	MHC class II T-A-b
6	85	88.5	93	2	T-cell receptor be
7	85	88.5	114	2	T-cell receptor be
8	85	88.5	115	2	T-cell receptor be
9	85	88.5	133	2	T-cell receptor be
10	81	84.4	114	2	T-cell receptor be
11	81	84.4	134	1	T-cell receptor be
12	81	84.4	145	2	T-cell receptor be
13	81	84.4	145	2	T-cell receptor be
14	80	83.3	114	2	T-cell receptor be
15	80	83.3	114	2	T-cell receptor be
16	80	83.3	115	2	T-cell receptor be
17	77	80.2	115	2	T-cell receptor be
18	74	77.1	38	2	T-cell receptor be
19	74	77.1	61	2	T-cell receptor be
20	74	77.1	79	2	T-cell receptor be
21	74	77.1	84	2	T-cell receptor be
22	74	77.1	91	2	T-cell receptor be
23	74	77.1	93	2	T-cell receptor be
24	74	77.1	101	2	T-cell receptor be
25	74	77.1	106	2	T-cell receptor be
26	74	77.1	114	2	T-cell receptor be
27	74	77.1	114	2	T-cell receptor be
28	74	77.1	115	2	T-cell receptor be
29	74	77.1	115	2	T-cell receptor be

30	74	77.1	115	2	S22038	T-cell receptor be
31	74	77.1	129	2	S57884	T-cell receptor be
32	74	77.1	132	2	S30441	T-cell receptor be
33	74	77.1	133	1	RWHU7B	T-cell receptor be
34	74	77.1	135	2	S38386	T-cell receptor be
35	74	77.1	135	2	S57882	T-cell receptor be
36	74	77.1	139	2	S38393	T-cell receptor be
37	74	77.1	141	2	S03495	T-cell receptor be
38	74	77.1	151	2	S34064	T-cell receptor be
39	73	76.0	102	2	S03492	T-cell receptor be
40	73	76.0	106	2	S25416	T-cell receptor be
41	73	76.0	113	2	S22039	T-cell receptor be
42	73	76.0	114	2	S17388	T-cell receptor be
43	73	76.0	114	2	S38313	T-cell receptor be
44	73	76.0	140	2	S36942	T-cell receptor be
45	72	75.0	114	2	G32537	T-cell receptor be

ALIGNMENTS

RESULT 1

138314 T-cell receptor beta chain V region (V-beta 8.1, germline) precursor - human (fr

C:Species: Homo sapiens (man)

C>Date: 16-Feb-1996 #sequence_revision 16-Feb-1996 #text_change 21-Jan-2000

C:Accession: 138314

R:SLIGHTOM, J.L.; Stenleniak, D.R.; Sleu, L.C.; Koop, B.F.; Hood, L.

Genomics 20, 149-168, 1994

A>Title: Nucleotide sequence analysis of 77.7 kb of the human V beta T-cell rece

A:Reference number: A54302; MUID:94292194

A:Accession: 138314

A:Status: Preliminary

A:Molecule type: DNA

A:Residues: 1-114 <RES>

A:Cross-references: EMBL:U03115; NID:g467918; PIDN:AA17713.1; PID:g467923

C:Genetics:

A:Gene: TCRBV851

A:Introns: 17/1

C:Superfamily: Immunoglobulin V region; Immunoglobulin homology

C:Keywords: T-cell receptor

F:35-113/Domain: Immunoglobulin homology <IMH>

Query Match 100.0%; Score 96; DB 2; Length 114;
Best Local Similarity 100.0%; Pred. No. 1.5e-08;
Matches 16; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

QY 1 CKPISGHNSLFWYRQT 16
DB 42 CKPISGHNSLFWYRQT 57

RESULT 2

S03510 T-cell receptor beta chain precursor V region (8.1) - human

C:Species: Homo sapiens (man)

C>Date: 30-Sep-1991 #sequence_revision 30-Sep-1991 #text_change 21-Jan-2000

C:Accession: S03510; S03512

R:Slu, G.; Strauss, E.C.; Lai, E.; Hood, L.E.

J. Exp. Med. 164, 1600-1614, 1986

A>Title: Analysis of a human V-beta gene subfamily.

A:Reference number: S03510; MUID:87035436

A:Accession: S03510

A:Status: translation not shown

A:Molecule type: DNA

A:Residues: 1-115 <STU>

A:Cross-references: EMBL:X07192

A>Note: This sequence was determined from the germline gene

Nucleic Acids Res. 15, 4991, 1987

A>Title: Germline sequence of two human T-cell receptor V-beta genes: V-beta-8.1

A:Reference number: S00520; MUID:8725979

A:Accession: S03512
 A:Molecule type: DNA
 A:Residues: 110 <SMI>
 A:Note: this sequence was determined from the germline gene
 C:Genetics:
 A:Introns: 17/1
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: T-cell receptor
 F:121/Domain: signal sequence #status predicted <SIG>
 F:22-115/Product: T-cell receptor beta chain (fragment) #status predicted <MAY>
 F:22-115/Domain: V region (V-beta 8.1) #status predicted <VRE>
 F:35-113/Domain: Immunoglobulin homology <IMM>

Query Match 100.0%; Score 96; DB 2; Length 115;
 Best Local Similarity 100.0%; Pred. No. 1.5e-08;
 Matches 16; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISCHNSLFMYROT 16
 |||
 Db 42 CKPISCHNSLFMYROT 57

RESULT 3

RMHUVY
 T-cell receptor beta chain precursor V region (Y735) - human
 N:Alternate names: T-cell receptor beta-1 chain J-B1.2 segment
 C:Species: Homo sapiens (man)
 C:Date: 03-Aug-1984 #sequence_rev1sion 03-Aug-1984 #text_change 22-Jun-1999
 C:Accession: A02000; E24687
 R:Yonaga, Y.; Yoshikata, Y.; Leggett, K.; Clark, S.P.; Aleksander, I.; Mak, T.W.
 Nature 308, 145-149, 1984
 A:Title: A human T cell-specific cDNA clone encodes a protein having extensive homology
 A:Reference number: A93324; MUID:84142269
 A:Accession: A02000
 A:Molecule type: mRNA
 A:Residues: 1-135 <YAN>
 A:Cross-references: GB:K01571
 A:Experimental source: clone Y735
 R:Yonaga, B.; Yoshikata, Y.; Vadasz, V.; Chin, B.; Mak, T.W.
 Proc. Natl. Acad. Sci. U.S.A. 82, 8624-8628, 1985
 A:Title: Organization and sequences of the diversity, joining, and constant region genes
 A:Reference number: A94081; MUID:86094276
 A:Accession: E24687
 A:Molecule type: DNA
 A:Residues: 121-135 <TOY>
 A:Cross-references: GB:M14158; NID:q338844; PIDN:AAA6069.1; PID:q553682
 C:Genetics:
 A:Gene: GDB:TCRB
 A:Cross-references: GDB:120405; OMIM:186930
 A:Map position: 7q35-7q35
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: glycoprotein; heterotrimer; receptor; T-cell
 F:1-21/Domain: signal sequence #status predicted <SIG>
 F:32-135/Product: T-cell receptor beta chain V region Y735 #status predicted <MAY>
 F:35-113/Domain: Immunoglobulin homology <IMM>
 F:42-111/Disulfide bonds: #status predicted
 F:90/Binding site: carbohydrate (Asn) (covalent) #status predicted

Query Match 100.0%; Score 96; DB 1; Length 135;
 Best Local Similarity 100.0%; Pred. No. 1.7e-08;
 Matches 16; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISCHNSLFMYROT 16
 |||
 Db 42 CKPISCHNSLFMYROT 57

RESULT 4

S57877
 T cell receptor CK14 beta chain - human (fragment)
 C:Species: Homo sapiens (man)

C:Date: 28-Oct-1995 #sequence_rev1sion 03-Nov-1995 #text_change 21-Jan-2000
 C:Accession: S57877
 R:Giegerich, G.; Pette, M.; Meln, E.; Eppien, J.T.; Wexler, H.; Hinkkanen, A.
 Eur. J. Immunol. 22, 753-758, 1992
 A:Title: Diversity of T cell receptor alpha and beta chain genes expressed by hu
 A:Reference number: S57869; MUID:92192091
 A:Accession: S57877
 A>Status: Preliminary
 A:Molecule type: mRNA
 A:Residues: 1-135 <GIE>
 A:Cross-references: EMBL:X58324; NID:q643002; PIDN:CAA41236.1; PID:q643003
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: T-cell receptor
 F:35-113/Domain: Immunoglobulin homology <IMM>

Query Match 100.0%; Score 96; DB 2; Length 135;
 Best Local Similarity 100.0%; Pred. No. 1.7e-08;
 Matches 16; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISCHNSLFMYROT 16
 |||
 Db 42 CKPISCHNSLFMYROT 57

RESULT 5

I71938
 MHC class II I-A-beta protein precursor - mouse (fragment)
 C:Species: Mus musculus (house mouse)
 C:Date: 02-Aug-1996 #sequence_rev1sion 02-Aug-1996 #text_change 21-Jan-2000
 C:Accession: I71938
 R:Spina, D.G.; Hansen, T.H.; Walsh, W.D.; Behlke, M.A.; Tillinghast, J.P.; Ch
 J. Immunol. 138, 3991-3995, 1987
 A:Title: Receptor diversity of Insulin-specific T cell lines from C57BL (H-2b) m
 A:Reference number: I55978; MUID:87224052
 A:Accession: I71938
 A>Status: Preliminary; translated from GB/EMBL/DBJ
 A:Molecule type: mRNA
 A:Residues: 1-134 <RES>
 A:Cross-references: GB:M16681; NID:q19473; PIDN:AAA39626.1; PID:q19474
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 F:35-113/Domain: Immunoglobulin homology <IMM>

Query Match 90.6%; Score 87; DB 2; Length 134;
 Best Local Similarity 81.2%; Pred. No. 5e-07;
 Matches 13; Conservative 2; Mismatches 1; Indels 0; Gaps 0;

OY 1 CKPISCHNSLFMYROT 16
 |||
 Db 42 CKPISCHNSLFMYROT 57

RESULT 6

S03488
 T-cell receptor beta chain precursor V region (clone HBP41) - human (fragment)
 C:Species: Homo sapiens (man)
 C:Date: 30-Jun-1992 #sequence_rev1sion 30-Jun-1992 #text_change 30-May-1997
 C:Accession: S03488
 R:Kimura, N.; Toyonaga, B.; Yoshikata, Y.; Triebel, F.; Debre, P.; Minden, M.D.;
 J. Exp. Med. 164, 739-750, 1986
 A:Title: Sequences and diversity of human T cell receptor beta chain variable reg
 A:Reference number: S03485; MUID:86306525
 A:Accession: S03488
 A:Molecule type: mRNA
 A:Residues: 1-93 <KIM>
 A:Cross-references: EMBL:X04925
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: T-cell receptor

Query Match 88.5%; Score 85; DB 2; Length 93;
 Best Local Similarity 87.5%; Pred. No. 7.4e-07;

Matches 14: Conservative 1; Mismatches 1; Indels 0; Gaps 0;
 OY 1 CKPISGHNSLFMYROT 16
 |||||||:|||||||
 Db 42 CKPISGHDLFWYROT 57

RESULT 7

138315
 T-cell receptor beta chain V region (V-beta 8.2, germline) precursor - human (fragment)
 C:Species: Homo sapiens (man)
 C>Date: 16-Feb-1996 #sequence_revision 16-Feb-1996 #text_change 21-Jan-2000
 C:Accession: I38315
 R:Slighlom, J.L.; Stemleniak, D.R.; Slew, L.C.; Koop, B.F.; Hood, L.
 Genomics 20, 149-168, 1994
 A:Title: Nucleotide sequence analysis of 77.7 kb of the human V beta T-cell receptor ger
 A:Reference number: A54502; MUID:94292194
 A:Accession: I38315
 A:Status: Preliminary
 A:Status: Preliminary
 A:Molecule type: DNA
 A:Residues: 1-114 <RES>
 A:Cross-references: EMBL:U03115; NID:9467918; PIDN:AAA17714.1; PID:9467924
 C:Genetics:
 A:Gene: TCRBV8S2
 A:Introns: 17/1
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: T-cell receptor
 F:35-113/Domain: Immunoglobulin homology <IMM>

Query Match

Best Local Similarity 88.5%; Score 85; DB 2; Length 114;
 Matches 14; Conservative 1; Mismatches 1; Indels 0; Gaps 0;

OY 1 CKPISGHNSLFMYROT 16
 |||||||:|||||||
 Db 42 CKPISGHDLFWYROT 57

RESULT 8

S03511
 T-cell receptor beta chain V region 8.2 precursor (clone H7.1 and others) - human
 C:Species: Homo sapiens (man)
 C>Date: 30-Sep-1991 #sequence_revision 30-Sep-1991 #text_change 21-Jan-2000
 C:Accession: S03511; S78510; S78511; S26270; S26271
 R:Stu, G.; Strauss, E.C.; Lal, E.; Hood, L.E.
 J. Exp. Med. 164, 1600-1614, 1986
 A:Title: Analysis of a human V-beta gene subfamily.
 A:Reference number: S03510; MUID:87035436
 A:Accession: S03511
 A:Status: translation not shown
 A:Molecule type: DNA
 A:Residues: 1-115 <STU>
 A:Cross-references: EMBL:X07222
 A:Experimental source: clone H7.1
 A:Genetics: G1
 A>Note: this sequence was determined from the germline gene
 R:Plaza, A.
 Submitted to the EMBL Data Library, February 1991
 A:Reference number: S78510
 A:Accession: S78510
 A:Molecule type: mRNA
 A:Residues: 1-114 <PLA>
 A:Cross-references: EMBL:X57619; NID:923934; PIDN:CAA40845.1; PID:923935
 A:Experimental source: clone HT2.12
 A:Genetics: G2
 A:Accession: S78511
 A:Molecule type: mRNA
 A:Residues: 1'D',3-114 <PLZ>
 A:Cross-references: EMBL:X57720; NID:923936; PIDN:CAA40887.1; PID:923937
 A:Experimental source: clone HT242
 A:Genetics: G3
 R:Plaza, A.; Kono, D.H.; Theofilopoulos, A.N.

J. Immunol. 147, 4360-4365, 1991
 A:Title: New human V-beta genes and polymorphic variants.
 A:Reference number: S26255; MUID:92091749
 A:Accession: S26270
 A:Molecule type: mRNA
 A:Residues: 1-96, 'R', '98-114 <PLM>
 A:Cross-references: EMBL:X57619
 A:Experimental source: clone HT2.12
 A:Genetics: G2
 A>Note: the authors translated the codon GGC for residue 2 as Asp
 A:Accession: S26271
 A:Molecule type: mRNA
 A:Residues: 1'D',3-96, 'R', '98-114 <PLF>
 A:Cross-references: EMBL:X57720
 A:Experimental source: clone HT242
 A:Genetics: G3
 A:Genetics: <G1>
 A:Gene: 8.2
 C:Genetics: <G2>
 A:Introns: 17/1
 A:Gene: 8.2a
 C:Genetics: <G3>
 A:Gene: 8.2b
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: T-cell receptor
 F:1-21/Domain: signal sequence #status predicted <SIG>
 F:22-115/Product: T-cell receptor beta chain (fragment) #status predicted <MAT>
 F:22-115/Domain: V region (V-beta 8.2) #status predicted <VRE>
 F:35-113/Domain: Immunoglobulin homology <IMM>

Query Match

Best Local Similarity 88.5%; Score 85; DB 2; Length 115;
 Matches 14; Conservative 1; Mismatches 1; Indels 0; Gaps 0;

OY 1 CKPISGHNSLFMYROT 16
 |||||||:|||||||
 Db 42 CKPISGHDLFWYROT 57

RESULT 9

S57870
 T cell receptor CK10 beta chain - human
 C:Species: Homo sapiens (man)
 C>Date: 27-Oct-1995 #sequence_revision 03-Nov-1995 #text_change 21-Jan-2000
 C:Accession: S57870
 R:Giegerich, G.; Petre, M.; Meini, E.; Epplen, J.T.; Wekerle, H.; Hinkkanen, A.
 Eur. J. Immunol. 22, 753-758, 1992
 A:Title: Diversity of T cell receptor alpha and beta chain genes expressed by hu
 A:Reference number: S57869; MUID:92192091
 A:Accession: S57870
 A:Status: preliminary
 A:Molecule type: mRNA
 A:Residues: 1-133 <GIE>
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: T-cell receptor
 F:35-113/Domain: Immunoglobulin homology <IMM>

Query Match

Best Local Similarity 88.5%; Score 85; DB 2; Length 133;
 Matches 14; Conservative 1; Mismatches 1; Indels 0; Gaps 0;

OY 1 CKPISGHNSLFMYROT 16
 |||||||:|||||||
 Db 42 CKPISGHNSLFMYROT 57

RESULT 10

A27553
 T-cell receptor beta chain precursor V region (VAK) - mouse
 C:Species: Mus musculus (house mouse)
 C>Date: 05-Jun-1988 #sequence_revision 05-Jun-1988 #text_change 23-Jul-1999

C:Accession: A27553
 Rieppien, J.T.; Bartels, F.; Becker, A.; Netz, G.; Prester, M.; Rinaldy, A.; Simon, M.M.
 Proc. Natl. Acad. Sci. U.S.A. 83, 4441-4445, 1986
 A:Title: Change in antigen specificity of cytotoxic T lymphocytes is associated with the
 A:Reference number: A27553; MUID:86533442
 A:Accession: A27553
 A:Molecule type: DNA
 A:Residues: 1-114 <EPP>
 A:Cross-references: GB:L29434; GB:M12775; GB:N00046; NID:9459887; PIDN:AAA40218.1; PID:9
 A:Note: this sequence was determined from the germline gene
 C:Comment: See entry PIR:A02002.
 C:Genetics:
 A:Introns: 17/1
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: T-cell receptor
 F:1-19/Domain: signal sequence #status predicted <SIG>
 F:20-114/Product: T-cell receptor beta chain V region VAK #status predicted <VAR>
 F:35-113/Domain: Immunoglobulin homology <IMM>

Query Match 84.4%; Score 81; DB 2; Length 114;
 Best Local Similarity 75.0%; Pred. No. 4e-06;
 Matches 12; Conservative 4; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGNSLFMYROT 16
 I:|||||:|||||
 DB 42 CEPISGSAVFMYROT 57

RESULT 11

RWMSB3
 T-cell receptor beta chain precursor V region (CTL-F3) - mouse
 C:Species: Mus musculus (house mouse)
 C:Date: 30-Jun-1987 #sequence_revision 30-Jun-1987 #text_change 30-May-1997
 C:Accession: A02002
 R:Chou, H.S.; Behtke, M.A.; Godambe, S.A.; Russell, J.H.; Brooks, C.G.; Loh, D.Y.
 EMBO J. 5, 2149-2155, 1986
 A:Title: T cell receptor genes in an alloreactive CTL clone: implications for rearrangement
 A:Reference number: A91048; MUID:87053852
 A:Accession: A02002
 A:Molecule type: mRNA
 A:Residues: 1-134 <CHO>
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: glycoprotein; heterotetramer; T-cell receptor
 F:1-19/Domain: signal sequence #status predicted <SIG>
 F:20-134/Product: T-cell receptor beta chain precursor V region (CTL-F3) #status predicted
 F:20-115/Region: V segment
 F:35-113/Domain: Immunoglobulin homology <IMM>
 F:116-119/Region: D segment
 F:120-134/Region: J segment
 F:42-111/Disulfide bonds: #status predicted
 F:90/Binding site: carbohydrate (Asn) (covalent) #status predicted

Query Match 84.4%; Score 81; DB 1; Length 134;
 Best Local Similarity 75.0%; Pred. No. 4.7e-06;
 Matches 12; Conservative 4; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGNSLFMYROT 16
 I:|||||:|||||
 DB 42 CEPISGSAVFMYROT 57

RESULT 12

S21651
 T-cell receptor beta chain - mouse
 C:Species: Mus musculus (house mouse)
 C:Date: 20-Feb-1995 #sequence_revision 20-Feb-1995 #text_change 23-Jul-1999
 C:Accession: S21651
 R:Yamagishi, H.
 submitted to the EMBL Data Library, November 1990
 A:Reference number: S21643
 A:Accession: S21651

A:Status: preliminary
 A:Molecule type: mRNA
 A:Residues: 1-145 <YAA>
 A:Cross-references: EMBL:X56708; NID:954647; PIDN:CAA40038.1; PID:954648
 C:Superfamily: Immunoglobulin V region; Immunoglobulin homology
 C:Keywords: T-cell receptor
 F:35-113/Domain: Immunoglobulin homology <IMM>

Query Match 84.4%; Score 81; DB 2; Length 145;
 Best Local Similarity 75.0%; Pred. No. 5.1e-06;
 Matches 12; Conservative 4; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGNSLFMYROT 16
 I:|||||:|||||
 DB 42 CEPISGSAVFMYROT 57

RESULT 13

RWMSB3
 T-cell receptor beta chain precursor (F5) - mouse
 C:Species: Mus musculus (house mouse)
 C:Date: 03-Aug-1984 #sequence_revision 01-Dec-2000 #text_change 01-Dec-2000
 C:Accession: S03716; S25118; A02134; A93336; B93333
 R:Palmer, M.S.; Bentley, A.; Gould, K.; Townsend, A.R.M.
 Nucleic Acids Res. 17, 2353, 1989
 A:Title: The T cell receptor from an influenza-A specific murine CTL clone.
 A:Reference number: S03715; MUID:89202046
 A:Accession: S03716
 A:Molecule type: mRNA
 A:Residues: 1-307 <PAL>
 A:Cross-references: EMBL:X14388; NID:954668; PIDN:CAA32563.1; PID:954669
 R:Austrup, F.; Kodell, V.; Kucharzik, T.; Kisch, E.
 submitted to the EMBL Data Library, July 1992
 A:Description: Characterization of idiotypic-specific I-Ed-restricted T suppressor
 /c mice.

A:Reference number: S25117
 A:Accession: S25118
 A:Status: preliminary
 A:Molecule type: mRNA

A:Residues: 1/'G'/'6', 'C', '8', 'V'/'F', '14', 'T', '16', 'N', '19', 'D', '21', '23', 'T', '25', '29', 'E', '31',
 'K', '86', 'E', '88', 'L', '90', 'S', '92', 'F', '94', '99', 'P', '101', 'E', '103', 'K', '105', '114', 'L', 'N', 'S', 'A', 'E', 'T', 'L', 'Y',
 A:Cross-references: EMBL:X67128; NID:954678; PIDN:CAA47607.1; PID:954679
 C:Superfamily: Immunoglobulin C region; Immunoglobulin homology
 C:Keywords: glycoprotein; heterotetramer; receptor; T-cell; transmembrane protein
 F:1-19/Domain: signal sequence #status predicted <SIG>
 F:20-307/Product: T-cell receptor beta chain #status predicted <MAT>
 F:158-228/Domain: Immunoglobulin homology <IMM>
 F:281-302/Domain: transmembrane #status predicted
 F:303-307/Domain: intracellular #status predicted <INT>

Query Match 84.4%; Score 81; DB 1; Length 307;
 Best Local Similarity 75.0%; Pred. No. 1.1e-05;
 Matches 12; Conservative 4; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGNSLFMYROT 16
 I:|||||:|||||
 DB 42 CEPISGSAVFMYROT 57

RESULT 14

S17379
 T-cell receptor beta chain V region (clone ICR02) - human (fragment)
 C:Species: Homo sapiens (man)
 C:Date: 25-Feb-1994 #sequence_revision 10-Nov-1995 #text_change 21-Jan-2000
 C:Accession: S17379
 R:Ferradini, L.; Roman-Roman, S.; Azocar, J.; Michalaki, H.; Trisbel, F.; Hercend
 Eur. J. Immunol. 21, 935-942, 1991
 A:Title: Studies on the human T cell receptor alpha/beta variable region genes. I
 A:Reference number: S17378; MUID:91209402
 A:Accession: S17379
 A:Status: preliminary

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OM protein - protein search, using sw model

Run on: February 9, 2002, 10:51:07 ; Search time 10.2 Seconds

(without alignments)
57.513 Million cell updates/sec

Title: US-09-591-789-1

Perfect score: 96

Sequence: 1 CKPISGHSLEFWYRQT 16

Scoring table: BLOSUM62

Searched: 100059 seqs, 3664827 residues

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

Database: SwissProt_39:*

Pred. No. is the number of results predicted by chance to have a
score greater than or equal to the score of the result being printed,
and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	Query Match	Length	ID	Description
1	96	100.0	135	TVB1_HUMAN	P01733 homo sapien
2	81	84.4	134	TVB7_MOUSE	P06320 mus musculu
3	74	77.1	133	TVB2_HUMAN	P04435 homo sapien
4	67	69.8	321	TCB_FLY	P11364 feline leuk
5	56	58.3	130	TVB8_MOUSE	P06321 mus musculu
6	56	58.3	133	TVB2_MOUSE	P01735 mus musculu
7	51	53.1	135	TVB1_MOUSE	P01734 mus musculu
8	47	49.0	122	TVB5_MOUSE	P04213 mus musculu
9	47	49.0	136	TVB4_MOUSE	P04212 mus musculu
10	47	49.0	137	Y137_ADE02	P03293 human adeno
11	47	49.0	567	NUZM_HUMAN	P48906 hansenula w
12	45	46.9	748	PHY1_SYNT3	O55168 synechocyst
13	44	45.8	120	TVB3_MOUSE	P01736 mus musculu
14	43	44.8	103	RPOM_ARCFU	O29033 archaeoglob
15	42	43.8	194	VA43_VACCC	P21065 vaccinia vl
16	42	43.8	194	VA43_VACCV	P26671 vaccinia vl
17	42	43.8	195	VA43_VARY	P33855 vaccinia vir
18	42	43.8	788	TRSI_HCMVA	P09695 human cytom
19	42	43.8	846	IRS1_HCMVA	P09715 human cytom
20	42	43.8	1129	PHYA_PETCR	P55141 petroselinu
21	41	42.7	108	RPOM_METJA	O58548 methanococc
22	41	42.7	492	CAT1_MAIZE	P18122 zea mays (m
23	41	42.7	540	YFE0_YEAST	P43562 saccharomyc
24	40.5	42.2	1183	YMK6_YEAST	P50942 saccharomyc
25	40	41.7	111	LV21_HUMAN	P01702 homo sapien
26	40	41.7	112	LV1B_HUMAN	P01700 homo sapien
27	40	41.7	242	GUB_BACSU	P04957 bacillus su
28	40	41.7	337	LEPB_HAESO	P36685 haemophilus
29	39	40.6	389	CHSB_PEA	P51082 pisum sativ
30	39	40.6	107	KV6E_MOUSE	P01679 mus musculu
31	39	40.6	288	YDIB_ECOLI	P28244 escherichia
32	39	40.6	323	A85B_MYCBO	P12942 mycobacteri
33	39	40.6	389	CHS1_PEA	O01286 pisum sativ

ALIGNMENTS

34	39	40.6	389	1	CHS2_PEA	O01287 pisum sativ
35	39	40.6	492	1	CAT1_HORVU	P55307 hordeum vul
36	39	40.6	492	1	CAT2_WHEAT	P55313 triticum ae
37	39	40.6	501	1	XYLB_LACIA	O9efg8 lactococcus
38	39	40.6	108	1	KVIC_HUMAN	P01595 homo sapien
39	38	39.6	111	1	LV2D_HUMAN	P01707 homo sapien
40	38	39.6	111	1	LV2F_HUMAN	P01709 homo sapien
41	38	39.6	115	1	KV5C_MOUSE	P01635 mus musculu
42	38	39.6	142	1	VPRL_MOUSE	P13372 mus musculu
43	38	39.6	142	1	VPR2_MOUSE	P13373 mus musculu
44	38	39.6	265	1	CHS7_MEDSA	P51080 medicago sa
45	38	39.6	285	1	CHS6_MEDSA	P51079 medicago sa

RESULT 1	TVB1_HUMAN	STANDARD:	PRT: 135 AA.
ID	TVB1_HUMAN		
AC	P01733:		
DT	21-JUL-1986 (Rel. 01, Created)		
DT	21-JUL-1986 (Rel. 01, Last sequence update)		
DT	15-JUL-1999 (Rel. 38, Last annotation update)		
DE	T-CELL RECEPTOR BETA CHAIN V REGION YT35 PRECURSOR.		
OS	Homo sapiens (Human).		
OC	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;		
OC	Mammalia; Eutheria; Primates; Catarrhini; Homiidae; Homo.		
OX	NCBI_TaxID=9606;		
RP	[1]		
RP	SEQUENCE FROM N.A.		
RX	MEDLINE=84142269; PubMed=6336315;		
RA	Yanagi Y., Yoshikai Y., Leggett K., Clark S.P., Alexander I.,		
RA	Mak T.W.;		
RT	"A human T cell-specific cDNA clone encodes a protein having		
RT	extensive homology to immunoglobulin chains.";		
RL	Nature 308:145-149(1984).		
DR	PIR: A02000; RWHUVY.		
DR	HSSP: P01789; 2MCP.		
DR	InterPro: IPR003599; Ig.		
DR	InterPro: IPR003006; Ig_MHC.		
DR	Pfam: PF00047; Ig: 1.		
DR	SMART: SM00409; IG: 1.		
KW	T-cell; Receptor; Signal.		
FT	SIGNAL	?	POTENTIAL.
FT	CHAIN	?	T-CELL RECEPTOR BETA CHAIN V REGION YT35.
FT	NON_TER	135	
FT	SEQUENCE	135 AA; 15097 MW; 9F8F913D33967CC9 CRC64;	

Query Match	100.0%;	Score 96;	DB 1;	Length 135;
Best Local Similarity	100.0%;	Pred. No. 2.5e-09;		
Matches 16; Conservative	0;	Mismatches	0;	Gaps 0;
OY	1 CKPISGHSLEFWYRQT 16			
DB	42 CKPISGHSLEFWYRQT 57			

RESULT 2	TVB7_MOUSE	STANDARD:	PRT: 134 AA.
ID	TVB7_MOUSE		
AC	P06320:		
DT	01-JAN-1988 (Rel. 06, Created)		
DT	01-JAN-1988 (Rel. 06, Last sequence update)		
DT	15-JUL-1999 (Rel. 38, Last annotation update)		
DE	T-CELL RECEPTOR BETA CHAIN V REGION CTL-F3 PRECURSOR.		
OS	Mus musculus (Mouse).		
OC	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;		
OC	Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.		
OX	NCBI_TaxID=10090;		
RP	[1]		
RP	SEQUENCE FROM N.A.		

RX MEDLINE-87053852; PubMed-3490968;
 RA Chou H.S., Behlke M.A., Godambe S.A., Russell J.H., Brooks C.G.,
 RA Loh D.Y.;
 RT "cell receptor genes in an allelic reactive CTL clone: implications for
 RT rearrangement and germine diversity of variable gene segments";
 RL EMBL J. 5:2149-2155(1986).
 DR PIR: A02002; R0003599;
 DR InterPro: IPR003599; Ig.
 DR Pfam: PF00047; Ig_1.
 DR SMART: SM00409; Ig_1.
 KM T-cell; Receptor; Glycoprotein; Signal.
 FT SIGNAL 1 19
 FT CHAIN 20 134 T-CELL RECEPTOR BETA CHAIN V REGION CTL-
 FT DOMAIN 20 115 F3.
 FT DOMAIN 116 119 V SEGMENT.
 FT DOMAIN 120 134 D SEGMENT.
 FT DISULFID 42 111 J SEGMENT.
 FT CARBOHYD 90 111 BY SIMILARITY.
 FT NON_TER 134 134 N-LINKED (GLCNAC. . .) (POTENTIAL).
 SQ SEQUENCE 134 AA; 14946 MW; C080FB24C81988F6 CRC64;

Query Match 84.48; Score 81; DB 1; Length 134;
 Best Local Similarity 75.08; Pred. No. 8.5e-07;
 Matches 12; Conservative 4; Mismatches 0; Indels 0; Gaps 0;

OY 1 CKPISGHNSLFMYROT 16
 ID 42 CDPISGHNSLFMYROT 57

RESULT 3
 TVB2_HUMAN STANDARD; PRT; 133 AA.
 AC P04435;
 DT 13-AUG-1987 (Rel. 05, Created)
 DT 13-AUG-1987 (Rel. 05, Last sequence update)
 DT 15-JUL-1999 (Rel. 38, Last annotation update)
 DE T-CELL RECEPTOR BETA CHAIN V REGION CTL-117 PRECURSOR.
 GN TCRB.
 OS Homo sapiens (Human).
 OC Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
 OC Mammalia; Eutheria; Primates; Catarrhini; Homindaes; Homo.
 OX NCBI_Taxid=9606;
 RN [1]
 RP SEQUENCE FROM N.A.
 X MEDLINE-86276770; PubMed-2426193;
 A Leiden J.M., Fraser J.D., Strominger J.L.;
 RT "The complete primary structure of the T-cell receptor genes from an
 RT allelic reactive cytotoxic human T-lymphocyte clone";
 RL Immunogenetics 24:17-23(1986).
 CC -1- MISCELLANEOUS: THIS SEQUENCE WAS DERIVED FROM A HUMAN CYTOTOXIC
 CC T-LYMPHOCYTE THAT IS T3+, T4+, T8-.
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 CC -----
 CC EMBL: M15564; AAA61027.1; -.
 DR PIR: A02001; RMH07B.
 DR InterPro: IPR003599; Ig.
 DR InterPro: IPR003599; Ig_MHC.
 DR Pfam: PF00047; Ig_1.
 DR SMART: SM00409; Ig_1.
 KM T-cell; Receptor; Glycoprotein; Signal.
 FT SIGNAL 1 21
 FT CHAIN 22 133 T-CELL RECEPTOR BETA CHAIN V REGION CTL-

FT DOMAIN 22 114 L17.
 FT DOMAIN 115 118 V SEGMENT.
 FT DOMAIN 119 133 D SEGMENT.
 FT CARBOHYD 30 30 J SEGMENT.
 FT CARBOHYD 37 37 N-LINKED (GLCNAC. . .).
 FT DISULFID 42 111 N-LINKED (GLCNAC. . .).
 FT NON_TER 133 133
 SQ SEQUENCE 133 AA; 14999 MW; 21030818D18D341F CRC64;

Query Match 77.18; Score 74; DB 1; Length 133;
 Best Local Similarity 75.08; Pred. No. 1.3e-05;
 Matches 12; Conservative 1; Mismatches 3; Indels 0; Gaps 0;

OY 1 CKPISGHNSLFMYROT 16
 ID 42 CDPISGHNSLFMYROT 57

RESULT 4
 TCB_FLV STANDARD; PRT; 321 AA.
 AC P11364;
 DT 01-JUL-1989 (Rel. 11, Created)
 DT 01-JUL-1989 (Rel. 11, Last sequence update)
 DT 15-JUL-1999 (Rel. 38, Last annotation update)
 DE T-CELL RECEPTOR BETA CHAIN T17T-22 PRECURSOR.
 GN V-TCR.
 OS Feline leukemia virus.
 OC Viruses; Retroviral viruses; Retroviridae; Gammaretrovirus.
 OX NCBI_Taxid=11768;
 RN [1]
 RP SEQUENCE FROM N.A.
 RX MEDLINE-87144638; PubMed-3029597;
 RA Fulton R., Forrest D., McFarlane R., Onions D., Nell J.C.;
 RT "Retroviral transduction of T-cell antigen receptor beta-chain and
 RT myc genes";
 RL Nature 326:190-194(1987).
 RN [2]
 RP REVISION TO 158-159.
 RA Fulton R.;
 RL Submitted (DEC-1987) to the EMBL/GenBank/DBJ databases.
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 CC -----
 CC EMBL: X05155; CAA28801.1; -.
 DR PIR: C26600; RMH07C.
 DR PIR: B26600; RMH07C.
 DR InterPro: IPR003599; Ig_MHC.
 DR InterPro: IPR003599; Ig_C1.
 DR InterPro: IPR003600; Ig_1like.
 DR Pfam: PF00047; Ig_2.
 DR SMART: SM00407; IgC1; 1.
 DR SMART: SM00410; Ig_1like; 1.
 KM T-cell; Receptor; Transmembrane; Glycoprotein; Signal.
 FT SIGNAL 1 28
 FT CHAIN 29 321 T-CELL RECEPTOR BETA CHAIN T17T-22.
 FT DOMAIN 29 122 V SEGMENT.
 FT DOMAIN 123 128 D SEGMENT.
 FT DOMAIN 129 144 J SEGMENT.
 FT DOMAIN 145 321 C REGION.
 SQ SEQUENCE 321 AA; 35581 MW; 11D2C3BF56811129 CRC64;

Query Match 69.88; Score 67; DB 1; Length 321;
 Best Local Similarity 73.38; Pred. No. 0.00048;

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 CC -----
 DR EMBL: J01917; NOT_ANNOTATED_CDS.
 DR PIR: A03865; A03865.
 DR Hypothetical protein.
 KW SEQUENCE 137 AA: 14356 MW: 87ACAG1AG3116856 CRC64:

Query Match 49.0% Score 47; DB 1; Length 137;
 Best Local Similarity 50.0% Pred. No. 0.47;
 Matches 6; Conservative 5; Mismatches 1; Indels 0; Gaps 0;

Oy 2 KPSGNSLFWY 13
 :|||||: 1;
 Db 60 EPVSGHSSVWV 71

RESULT 11
 NU2M_HANNI STANDARD: PRT: 567 AA.
 AC P48906; 01-FEB-1996 (Rel. 33, Created)
 DT 01-FEB-1996 (Rel. 33, Last sequence update)
 DT 15-JUL-1999 (Rel. 38, Last annotation update)
 DE NAOH-UBIQUINONE OXIDOREDUCTASE CHAIN 2 (EC 1.6.5.3).
 GN ND2.
 OS Hansenula wingel (Yeast).
 OC Mitochondrion.
 OC Eukaryota; Fungi; Ascomycota; Saccharomycotina; Saccharomycetes;
 OC Saccharomycetales; Saccharomycetaceae; Pichia.
 OX NCBI_TaxID=4907;
 RN [1]
 RP SEQUENCE FROM N.A.
 RC STRAIN=21;
 RA Sekito T., Okamoto K., Kitano H., Yoshida K.;
 RT "Yeast Hansenula wingel mitochondria genome's complete DNA sequence
 RT demonstrated unique characteristics";
 RL Nucleic Acids Symp. Ser. 31:233-234(1994).
 CC -1- CATALYTIC ACTIVITY: NADH + UBIQUINONE -> NAD(+) + UBIQUINOL.
 CC -1- SUBCELLULAR LOCATION: INTEGRAL MEMBRANE PROTEIN. MITOCHONDRIAL
 CC INNER MEMBRANE.
 CC -1- SIMILARITY: BELONGS TO THE COMPLEX I SUBUNIT 2 FAMILY.
 CC -----
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 CC -----
 DR EMBL: D31785; BAA06573.1; -
 DR InterPro: IPR001750; Oxidored_g1.
 DR Pfam: PF00361; oxidored_g1.1.
 KW Oxidoreductase; NAD; Ubiquinone; Mitochondrion; Transmembrane.
 SO SEQUENCE 567 AA: 66958 MW: 68C0F09B8FB012BE CRC64:

Query Match 49.0% Score 47; DB 1; Length 567;
 Best Local Similarity 88.9% Pred. No. 2.1;
 Matches 8; Conservative 1; Mismatches 0; Indels 0; Gaps 0;

Oy 5 SGHNSLFWY 13
 :|||||: 1;
 Db 184 SGHNSLFWY 192

RESULT 12
 PHY1_SYNV3

ID PHY1_SYNV3 STANDARD: PRT: 748 AA.
 AC O55168;
 DT 01-NOV-1997 (Rel. 35, Created)
 DT 01-NOV-1997 (Rel. 35, Last sequence update)
 DT 20-AUG-2001 (Rel. 40, Last annotation update)
 DE PHYTOCHROME-LIKE PROTEIN CPH1 (EC 2.7.3.-) (LIGHT-REGULATED HISTIDINE
 DE KINASE 1) (BACTERIOPHYTOCHROME CPH1).
 GN CPH1 OR SLR0473.
 OS Synechocystis sp. (strain PCC 6803).
 OC Bacteria; Cyanobacteria; Chroococcales; Synechocystis.
 OX NCBI_TaxID=1148;
 RN [1]
 RP SEQUENCE FROM N.A.
 RP MEDLINE=96127529; PubMed=8590279;
 RA Kaneko T., Tanaka A., Sato S., Kotani H., Sazuka T., Miyajima N.,
 RA Sugita M., Tabata S.;
 RT "Sequence analysis of the genome of the unicellular cyanobacterium
 RT Synechocystis sp. strain PCC6803. 1. Sequence features in the 1 Mb
 RT region from map positions 648 to 928 of the genome";
 RL DNA Res. 2:153-166(1995).
 RN [2]
 RP CHARACTERIZATION, AND MUTAGENESIS OF HIS-260.
 RP MEDLINE=97426627; PubMed=9278513;
 RA Yeh K.-C., Wu S.-H., Murphy J.T., Lagarias J.C.;
 RT "A cyanobacterial phytochrome two-component light sensory system";
 RL Science 277:1505-1508(1997).
 RN [3]
 RP CHARACTERIZATION.
 RP MEDLINE=20290819; PubMed=10828948;
 RA Park C.-M., Shim J.-Y., Yang S.-S., Kang J.-G., Kim J.-I., Luka Z.,
 RA Song P.-S.;
 RT "Chromophore-apoprotein interactions in Synechocystis sp. PCC6803
 RT phytochrome Cph1.";
 RL Biochemistry 39:6349-6356(2000).
 CC -1- FUNCTION: REGULATOR PHOTORECEPTOR WHICH EXISTS IN TWO FORMS THAT
 CC ARE REVERSIBLY INTERCONVERTIBLE BY LIGHT: THE R FORM THAT ABSORBS
 CC MAXIMALLY IN THE RED REGION OF THE SPECTRUM AND THE FR FORM THAT
 CC ABSORBS MAXIMALLY IN THE FAR-RED REGION. HAS ALSO A SLIGHT BLUE
 CC SHIFT FOR THE FAR-RED MAXIMUM. FORMS A TWO-COMPONENT SYSTEM WITH
 CC THE RCP1 RESPONSE REGULATOR.
 CC -1- SUBUNIT: HOMODIMER.
 CC -1- PTM: CONTAINS ONE COVALENTLY LINKED TETRAPYRROLE CHROMOPHORE (BY
 CC SIMILARITY).
 CC -1- MISCELLANEOUS: THE R FORM EXHIBITS BOTH ATP-DEPENDENT
 CC AUTOPHOSPHORYLATION AND PHOSPHOTRANSFER TO RCP1 ACTIVITIES. UNLIKE
 CC THE HIGHER PLANTS WHERE PER IS THOUGHT TO BE THE ACTIVE FORM.
 CC -1- SIMILARITY: IN THE N-TERMINAL SECTION; BELONGS TO THE PHYTOCHROME
 CC FAMILY.
 CC -1- SIMILARITY: IN THE C-TERMINAL SECTION; TO OTHER PROKARYOTIC
 CC SENSOR TRANSDUCTION HISTIDINE KINASES.
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 CC -----
 DR EMBL: D64001; BAA10307.1; -
 DR InterPro: IPR000410; Bctrl_sensor.
 DR InterPro: IPR003018; GAF.
 DR InterPro: IPR003594; HATPase_C.
 DR InterPro: IPR003661; His_KinA.
 DR InterPro: IPR000014; PAS.
 DR InterPro: IPR001294; Phytochrome.
 DR Pfam: PF01590; GAF.1.
 DR Pfam: PF02518; HATPase_C.1.
 DR Pfam: PF00360; phytochrome.1.
 DR Pfam: PF00512; signal.1.
 DR SMART: SM00065; GAF.1.
 DR SMART: SM00387; HATPase_C.1.
 DR SMART: SM00388; HisKA.1.

DR SMART: SM00091; PAS: 1.
 DR PROSITE: PS00245; PHYTOCHROME_1; FALSE_NEG.
 DR PROSITE: PS50046; PHYTOCHROME_2; 1.
 KW Sensory transduction; Transferrase; Kinase; Phosphorylation;
 KW Chromophore; Phytochrome; Chromophore; Complete proteome.
 FT DOMAIN 20 510 CHROMOPHORE BINDING DOMAIN.
 FT BINDING 511 748 SENSORY KINASE TRANSMITTER DOMAIN.
 FT MOD_RES 260 260 CHROMOPHORE (BY SIMILARITY).
 FT MUTAGEN 538 538 PHOSPHORYLATION (AUTO-) (BY SIMILARITY).
 FT H->K: NO AUTOPHOSPHORYLATION; NO
 PHOSPHOTRANSFER TO RCP1.
 SO SEQUENCE 748 AA: 84232 MW: A9ECA6D8DB3C68A CRC64;

Query Match 46.9%; Score 45; DB 1; Length 748;
 Best Local Similarity 58.3%; Pred. No. 6;
 Matches 7; Conservative 2; Mismatches 3; Indels 0; Gaps 0;

OY 3 PISGHSLSFWYR 14
 11: 11 11
 D 430 PIARHNLMLFR 441

RESULT 13
 TVB3_MOUSE
 ID TVB3_MOUSE STANDARD: PRT: 120 AA.
 AC P01736;
 DT 21-JUL-1986 (Rel. 01, Created)
 DT 21-JUL-1986 (Rel. 01, Last sequence update)
 DT 15-JUL-1998 (Rel. 38, Last annotation update)
 DE T-CELL RECEPTOR BETA CHAIN V REGION PHDS203 PRECURSOR (FRAGMENT).
 OS Mus musculus (Mouse).
 OC Eukaryota; Metazoa; Chordata; Vertebrata; Euteleostomi;
 OC Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.
 OX NCBI_TaxID=10090;
 RN [1]
 RP SEQUENCE FROM N.A.
 RC STRAIN-BALB.B;
 RX MEDLINE=84245824; PubMed=6310561;
 RA Salto H., Kranz D.M., Takagaki Y., Hayday A.C., Eisen H.N.,
 RA Tonggava S.;
 RT "Complete primary structure of a heterodimeric T-cell receptor
 deduced from cDNA sequences.";
 RL Nature 309:757-762(1984).
 CC CC
 CC -1- MISCELLANEOUS: THIS CLONE WAS ISOLATED FROM A CYTOTOXIC T
 LYMPHOCYTE.
 DR PIR: A02006; R0MSV2.
 DR InterPro: IPR003592; IG.
 DR InterPro: IPR003006; IG_MHC.
 DR Pfam: PF00047; IG_1.
 DR SMART: SM00409; IG_1.
 FT T-cell: Receptor; Signal.
 FT NON_TER 1 1
 FT SIGNAL <1 11
 FT CHAIN 12 120 T-CELL RECEPTOR BETA CHAIN V REGION
 FT DOMAIN 12 106 PHDS203.
 FT 120 120 V SEGMENT.
 FT DISULFID 34 102 J SEGMENT.
 FT NON_TER 120 120 BY SIMILARITY.
 SO SEQUENCE 120 AA: 13670 MW: D6B1011504969DCE CRC64;

Query Match 45.8%; Score 44; DB 1; Length 120;
 Best Local Similarity 40.0%; Pred. No. 1.3;
 Matches 6; Conservative 3; Mismatches 6; Indels 0; Gaps 0;

OY 1 CRPISGHSLSFWYRQ 15
 1 111 111
 DB 34 CGODMSHETMYWYRQ 48

RESULT 14

FROM_ARCFU
 ID FROM_ARCFU STANDARD: PRT: 103 AA.
 AC O29033;
 DT 15-JUL-1998 (Rel. 36, Created)
 DT 15-JUL-1998 (Rel. 36, Last sequence update)
 DT 20-AUG-2001 (Rel. 40, Last annotation update)
 DE DNA-DIRECTED RNA POLYMERASE SUBUNIT M (EC 2.7.7.6).
 GN RPOM OR AFI235.
 OS Archaeoglobus fulgidus.
 OC Archaea; Euryarchaeota; Archaeoglobales; Archaeoglobaceae;
 OC Archaeoglobus.
 OX NCBI_TaxID=2234;
 RN [1]
 RP SEQUENCE FROM N.A.
 RC STRAIN-VC-16 / DSM 4304 / ATCC 49558;
 RX MEDLINE=98049343; PubMed=9389475;
 RA Kleink H.-P., Clayton R.A., Tomb J.-F., White O., Nelson K.E.,
 RA Ketchum K.A., Dodson R.J., Gwyn M., Hickey E.K., Peterson J.D.,
 RA Richardson D.L., Kerlavage A.R., Graham D.E., Kyriades N.C.,
 RA Fleischmann R.D., Quackenbush J., Lee N.H., Sutton G.G., Gill S.,
 RA Kirschner E.F., Dougherty B.A., McKenney K., Adams M.D., Loftus B.,
 RA Peterson S., Reich C.I., McNeil L.K., Badger J.H., Glodek A., Zhou L.,
 RA Overbeek R., Gocayne J.D., Weidman J.F., McDonald L., Utterback T.,
 RA Cotton M.D., Spriggs T., Artlich P., Kalne B.P., Sykes S.M.,
 RA Sadow P.W., D'Andrea K.P., Bowman C., Fujii C., Garland S.A.,
 RA Mason T.M., Olsen G.J., Fraser C.M., Smith H.O., Woese C.R.,
 RA Venter J.C.;
 RT "The complete genome sequence of the hyperthermophilic, sulphate-
 reducing archaeon Archaeoglobus fulgidus.";
 RL Nature 390:364-370(1997).
 CC CC
 CC -1- FUNCTION: DNA-DEPENDENT RNA POLYMERASE CATALYZES THE TRANSCRIPTION
 OF DNA INTO RNA USING THE FOUR RIBONUCLEOSIDE TRIPHOSPHATES AS
 SUBSTRATES.
 CC CC
 CC -1- CATALYTIC ACTIVITY: N NUCLEOSIDE TRIPHOSPHATE - N PYROPHOSPHATE +
 RNA(N).
 CC CC
 CC -1- SIMILARITY: BELONGS TO THE ARCHAEABACTERIA RPOM / EUKARYOTIC RPA12/
 RPB9 / RPB11 RNA POLYMERASE FAMILY.
 CC CC
 CC -1- SIMILARITY: BELONGS TO THE TRANSCRIPTION FACTOR S-II FAMILY.
 CC CC
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 DR EMBL: AE001019; AAB90009.1; -.
 DR HSSP: Q56254; 10YP.
 DR TIGR: AF1215; -.
 DR InterPro: IPR001529; RNA_POL_M_15KD.
 DR InterPro: IPR001222; znf_TFIS.
 DR Pfam: PF02150; RNA_POL_M_15KD; 1.
 DR Pfam: PF01096; TFIS; 1.
 DR SMART: SM00440; znf_C2C2; 1.
 DR PROSITE: PS00466; TFIS; 1.
 DR PROSITE: PS01030; RNA_POL_M_15KD; 1.
 KW Transferrase; DNA-directed RNA polymerase; Transcription; Zinc-finger;
 KW Complete proteome.
 FT ZN_FING 4 23 C4-TYPE.
 FT ZN_FING 64 95 ZN-RIBBON.
 SO SEQUENCE 103 AA: 12057 MW: 4C189F2AC1CB62B CRC64;

Query Match 44.8%; Score 43; DB 1; Length 103;
 Best Local Similarity 54.5%; Pred. No. 1.6;
 Matches 6; Conservative 1; Mismatches 4; Indels 0; Gaps 0;

OY 3 PISGHSLSFWY 13
 1 111 111
 DB 65 PACGHNEAFWM 75

```

RESULT 15
VA43_VACCC STANDARD: PRT: 194 AA.
ID VA43_VACCC
AC P21065;
DT 01-FEB-1991 (Rel. 17, Created)
DT 01-FEB-1991 (Rel. 17, Last sequence update)
DT 20-AUG-2001 (Rel. 40, Last annotation update)
DE PROTEIN A43.
CN A43R.
OS Vaccinia virus (strain Copenhagen).
OC Viruses: dsDNA viruses, no RNA stage: Poxviridae: Chordopoxvirinae:
OC Orthopoxvirus.
OX NCBI_TaxID=10249;
RN [1]
RP SEQUENCE FROM N.A.
RX MEDLINE=91021027; PubMed=2219722;
RA Goebel S.J., Johnson G.P., Perkus M.E., Davis S.W., Winslow J.P.,
RA Paoletti E.;
RT "The complete DNA sequence of vaccinia virus.";
RL Virology 179:247-266(1990).
N [2]
RP COMPLETE GENOME.
RA Goebel S.J., Johnson G.P., Perkus M.E., Davis S.W., Winslow J.P.,
RA Paoletti E.;
RT "Appendix to 'The complete DNA sequence of vaccinia virus.'";
RL Virology 179:517-563(1990).
CC .....
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CC .....
DR EMBL: M35027; AAA48174.1; -
DR PIR: I42521.
SO SEQUENCE 194 AA: 22635 MW: 639990229C05591F CRC64:

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Query Match 43.88; Score 42; DB 1; Length 194;
Best Local Similarity 42.98; Pred. No. 4.7;
Matches 6; Conservative 4; Mismatches 4; Indels 0; Gaps 0;
QY 1 CKPISGHNSLFWYR 14
DB 144 CITIIGYDSIIWYK 157

```

Search completed: February 9, 2002, 10:54:12
 Job time: 185 sec

GenCore version 4.5
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OK protein - protein search, using sw model

Run on: February 9, 2002, 10:50:37 ; Search time 23 Seconds

(without alignments)
101.755 Million cell updates/sec

Title: US-09-591-789-1

Perfect score: 96

Sequence: 1 CKPISGHSLSFYRQT 16

Scoring table: BLOSUM62

Gapop 10.0 , Gapext 0.5

Searched: 473505 seqs, 146272329 residues

Total number of hits satisfying chosen parameters: 473505

Minimum DB seq length: 0

Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%

Maximum Match 100%

Listing first 45 summaries

Database :

1: SP archaea: *
2: SP bacteria: *
3: SP fungi: *
4: SP human: *
5: SP invertebrate: *
6: SP mammal: *
7: SP mhc: *
8: SP organelle: *
9: SP phage: *
10: SP plant: *
11: SP rodent: *
12: SP virus: *
13: SP vertebrate: *
14: SP unclassified: *

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	Query Match	Length	DB ID	Description
1	56	58.3	127	11	09D3G4
2	47	49.0	615	11	088576
3	47	49.0	615	11	088576
4	47	49.0	615	11	088576
5	45	46.9	421	10	098Y90
6	44.5	46.4	981	5	09NM63
7	44	45.8	215	11	09D2M6
8	44	45.8	234	11	099N04
9	44	45.8	428	2	09L2D5
10	44	45.8	556	10	09LPM2
11	43	44.8	235	13	090770
12	43	44.8	331	12	098337
13	43	44.8	381	10	094013
14	43	44.8	504	8	09GPF6
15	43	44.8	504	8	09GPF9
16	43	44.8	504	8	09GPF8
17	43	44.8	504	8	09GPF4
18	43	44.8	504	8	09GPF4
19	43	44.8	504	8	09GPF4

RESULT	ID	PRELIMINARY	PRT	127 AA	ALIGNMENTS
1	09D3G4	09D3G4			
AC	01-JUN-2001 (TREMBLrel. 17, Created)				
DT	01-JUN-2001 (TREMBLrel. 17, Last sequence update)				
DT	01-JUN-2001 (TREMBLrel. 17, Last annotation update)				
DE	5830405F06RIK PROTEIN.				
GN	5830405F06RIK.				
OS	Mus musculus (Mouse).				
OC	Eukaryota; Metazoa; Chordata; Vertebrata; Euteleostomi;				
OC	Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.				
OX	NCBI_TaxID=10090;				
RN	[1]				
RP	SEQUENCE FROM N.A.				
RC	STRAIN=C57BL/6J; TISSUE=THYMUS;				
RX	MEDLINE=21085660; PubMed=11217851;				
RA	Kawai J., Shinagawa A., Shibata K., Yoshino M., Itoh M., Ishii Y.,				
RA	Atakawa T., Hara A., Fukunishi Y., Konno H., Adachi J., Fukuda S.,				
RA	Alzawa K., Izawa M., Nishi K., Kiyosawa H., Kondo S., Yamataka I.,				
RA	Saito T., Okazaki Y., Gojohori T., Bono H., Kasukawa T., Saito R.,				
RA	Kadota K., Matsuda H.A., Ashburner M., Batalov S., Casavant T.,				
RA	Fleischmann W., Gaasterland T., Gissi C., King B., Kochiya H.,				
RA	Kuehl P., Lewis S., Matsuo Y., Nikaido I., Pesole G., Quackenbush J.,				
RA	Schriml L.M., Staudt F., Suzuki R., Tomita M., Wagner L., Watanabe T.,				
RA	Sakai K., Okido T., Furuno M., Aono H., Baldarelli R., Barsh G.,				
RA	Blake J., Boffelli D., Bojunga N., Carninci P., de Bonaldo M.F.,				
RA	Brownstein M.J., Bult C., Fletcher C., Fujita M., Gariboldi M.,				
RA	Guinacchini S., Hill D., Hofmann M., Hume D.A., Kamliya M., Lee N.H.,				
RA	Lyons P., Marchionni L., Mashima J., Marzelli J., Mombere P.,				
RA	Nordone P., Ring B., Ringwald M., Rodriguez I., Sakamoto N.,				
RA	Sasaki H., Sato K., Schoenbach C., Seya T., Shibata Y., Storch K.-F.,				
RA	Suzuki H., Toyooka K., Wang K.H., Weitz C., Whitaker C., Wilmink L.,				
RA	Wynshaw-Boris A., Yoshida K., Hasegawa Y., Kawai H., Kohsaki S.,				
RA	Hayashizaki Y.;				
RT	"Functional annotation of a full-length mouse cDNA collection.";				
RL	Nature 409:685-690(2001).				
CC	DOMAIN.				
CC	EMBL: AK017898; BAB30995.1; "				
DR	EMBL: AK017898; BAB30995.1; "				
DR	EMBL: MGI:1923265; 5830405F06RIK.				
DR	InterPro: IPR003599; Ig.				

DB 154 SGTVSFMYROT 165

RESULT 4

09FTF3 PRELIMINARY: PRT: 621 AA.

ID 09FTF3:

AC 01-MAR-2001 (TREMBLrel. 16, Created)

DT 01-MAR-2001 (TREMBLrel. 16, Last sequence update)

DT 01-JUN-2001 (TREMBLrel. 17, Last annotation update)

DE PUTATIVE RECEPTOR KINASE.

GN P0463F06.16 OR OJ1212.B09.24.

OS Oryza sativa (Rice).

OC Eukaryota: Viridiplantae: Streptophyta: Embryophyta: Tracheophyta:

OC Spermatophyta: Magnoliophyta: Liliopsida: Poales: Poaceae;

OC Ehrhartoideae: Oryzaceae; Oryza.

NCBI_TaxID=4530;

(1)

RP SEQUENCE FROM N.A.

RC STRAIN-CV. NIPPONBARE.

RA Sasaki T., Matsumoto T., Yamamoto K.;

RT "Oryza sativa nipponbare(GA3) genomic DNA, chromosome 1, PAC

clone: P0463F06."

RL Submitted (SEP-2000) to the EMBL/Genbank/DBJ databases.

RN [2]

RP SEQUENCE FROM N.A.

RC STRAIN-CV. NIPPONBARE;

RA Sasaki T., Matsumoto T., Yamamoto K.;

RT "Oryza sativa nipponbare(GA3) genomic DNA, chromosome 1, BAC

clone: OJ1212.B09."

RL Submitted (FEB-2001) to the EMBL/Genbank/DBJ databases.

DR EMBL: AP002867; BAB17126.1; -

DR EMBL: AP003338; BAB39451.1; -

DR InterPro: IPR000719; Euk_pkinase.

DR InterPro: IPR003015; HLH_MYC.

DR InterPro: IPR001245; Tyr_kin.

DR Pfam: PF00069; pkinase; 1.

DR SMART: SM00221; STYKC; 1.

DR SMART: SM00220; S_TKC; 1.

DR SMART: SM00219; TYKIC; 1.

DR PROSITE: PS00038; HELIX_LOOP_HELIX; UNKNOWN_1.

DR PROSITE: PS50011; PROTEIN_KINASE_DOM; 1.

DR ATP-binding; Kinase; Receptor; Transferase.

KW SEQUENCE 621 AA; 69776 MW; 8D24112786B99EF CRC64;

SO Query Match 49.0%; Score 47; DB 10; Length 621;

Best Local Similarity 69.2%; Pred. No. 8.6;

Matches 9; Conservative 1; Mismatches 1; Indels 2; Gaps 1;

OY 3 PIS-CHNSLFWY 13

DB 153 PISCLGHNNQFWY 165

RESULT 5

09SY90 PRELIMINARY: PRT: 421 AA.

ID 09SY90:

AC 01-MAY-2000 (TREMBLrel. 13, Created)

DT 01-MAY-2000 (TREMBLrel. 13, Last sequence update)

DT 01-MAY-2000 (TREMBLrel. 13, Last annotation update)

DE T25824.5 PROTEIN.

GN T25824.5.

OS Arabidopsis thaliana (Mouse-ear cress).

OC Eukaryota: Viridiplantae: Streptophyta: Tracheophyta:

OC Spermatophyta: Magnoliophyta: eudicotyledons; core eudicots; Rosidae;

OC eurosids II; Brassicales; Brassicaceae; Arabidopsis.

NCBI_TaxID=3702;

(1)

RP SEQUENCE FROM N.A.

RC STRAIN-CV. COLUMBIA;

RA Federspiel N.A., Palm C.J., Conway A.B., Conn L., Hansen N.F.,
RA Altafi H., Araujo R., Huizer L., Rowley D., Buehler E., Dunn P.,
RA Gonzalez A., Kremenetskaya I., Kim C., Lenz C., Li J., Liu S.,
RA Luros S., Schwartz J., Shinn P., Tortum M., Vysotskaya V.S.,
RA Walker M., Yu G., Ecker J., Theologis A., Davis R.W.,
RL Submitted (APR-1999) to the EMBL/Genbank/DBJ databases.
DR EMBL: AC005850; AAD25550.1; -

SO SEQUENCE 421 AA; 48772 MW; 5B1ADF00118431E CRC64;

Query Match 46.9%; Score 45; DB 10; Length 421;

Best Local Similarity 60.0%; Pred. No. 13;

Matches 6; Conservative 1; Mismatches 3; Indels 0; Gaps 0;

OY 6 CHNSLFWYRQ 15

DB 301 GHGCFYWRQ 310

RESULT 6

09NN63 PRELIMINARY: PRT: 981 AA.

ID 09NN63:

AC 01-OCT-2000 (TREMBLrel. 15, Created)

DT 01-OCT-2000 (TREMBLrel. 15, Last sequence update)

DT 01-JUN-2001 (TREMBLrel. 17, Last annotation update)

DE POSSIBLE HYPOTHEICAL 46.8 KDA PROTEIN IN CLCT1-PDS2 INTERGENIC REGION

(FRAGMENT).

GN LM15.368.

OS Leishmania major.

OC Eukaryota; Euklenozoa; Kinetoplastida; Trypanosomatidae; Leishmania.

NCBI_TaxID=5664;

(1)

RP SEQUENCE FROM N.A.

RC STRAIN-FRIEDLIN;

RA Murphy L., Quail M., Harris D., Rajandream M., Ivens A., Barrell B.;

RL Submitted (JUL-2000) to the EMBL/Genbank/DBJ databases.

DR EMBL: AL160371; CAC00347.1; -

DR InterPro: IPR000613; Pseudou_synth.

DR InterPro: IPR002990; PSI_RLU.

DR Prodom: PD001819; Pseudou_synth; 1.

DR PROSITE: PS01129; PSI_RLU; 1.

FT NON_TER 1

FT NON_TER 981

SO SEQUENCE 981 AA; 103596 MW; 93B5C673E15204D2 CRC64;

Query Match 46.4%; Score 44.5; DB 5; Length 981;

Best Local Similarity 53.3%; Pred. No. 36;

Matches 8; Conservative 2; Mismatches 2; Indels 3; Gaps 1;

OY 1 CKPI---SGHNSLFW 12

DB 696 CRPGCLDHNHNSLFW 710

RESULT 7

09D2W6 PRELIMINARY: PRT: 215 AA.

ID 09D2W6:

AC 01-JUN-2001 (TREMBLrel. 17, Created)

DT 01-JUN-2001 (TREMBLrel. 17, Last sequence update)

DT 01-JUN-2001 (TREMBLrel. 17, Last annotation update)

DE 913042211ORIK PROTEIN.

GN 913042211ORIK.

OS Mus musculus (Mouse).

OC Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;

OC Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Mus.

NCBI_TaxID=10090;

(1)

RP SEQUENCE FROM N.A.

RC STRAIN-C57BL/6J; TISSUE=CECUM;

RX MEDLINE=21085660; PubMed=11217851;

RA Kawai J., Shlnagawa A., Shibata K., Yoshino M., Itoh M., Ishii Y.,
 RA Atakawa T., Hara A., Fukunishi Y., Kono H., Adachi J., Fukuda S.,
 RA Aizawa K., Izawa M., Nishi K., Kiyosawa H., Kondo S., Yamataka I.,
 RA Saito T., Okazaki Y., Gojobori T., Bono H., Kasukawa T., Saito R.,
 RA Kadota K., Matsuda H.A., Ashburner M., Batalov S., Casavant T.,
 RA Fleischmann W., Gaasterland T., Gissi C., King B., Kochiwa H.,
 RA Knehl P., Lewis S., Matsuo Y., Nikaido I., Pesole G., Quackenbush J.,
 RA Schirral L.M., Staudil F., Suzuki R., Tomita M., Wagner L., Washio T.,
 RA Sakai K., Okido T., Furuno M., Aono H., Baldarelli R., Barsh G.,
 RA Blake J., Boffelli D., Bojunga N., Carlini P., de Bonaldo M.F.,
 RA Brownstein M.J., Bult C., Fletcher C., Fujita M., Gariboldi M.,
 RA Gusticich S., Hill D., Hofmann M., Hume D.A., Kamiya M., Lee N.H.,
 RA Lyons P., Marchionni L., Mashima J., Mazzarelli J., Mombaerts P.,
 RA Nordone P., Ring B., Ringwald M., Rodriguez I., Sakamoto N.,
 RA Sasaki H., Sato K., Schoenbach C., Seya T., Shibata Y., Storch K.-F.,
 RA Suzuki H., Toyooka K., Wang K.H., Wetz C., Whitaker C., Wilming L.,
 RA Wyshew-Boris A., Yoshida K., Hasegawa Y., Kawai H., Kohsaki S.,
 RA Hayashizaki Y.,
 RT "Functional annotation of a full-length mouse cDNA collection.",
 RL Nature 409:685-690(2001).
 JR EMBL: AK018686; BAB3134.1;
 CR MGI:1918846; 9130422110R1K.
 SO SEQUENCE 215 AA; 23722 MW; 3C6B3C964E18ED3E CRC64;

Query Match 45.8%; Score 44; DB 11; Length 215;
 Best Local Similarity 41.7%; Pred. NO. 9.6;
 Matches 5; Conservative 7; Mismatches 0; Indels 0; Gaps 0;

OY 4 ISGNLSLFMYRO 15

DB 176 LAGYSSIFMWKQ 187

RESULT 8
 O99N04 PRELIMINARY; PRT; 234 AA.
 DT 01-JUN-2001 (Tremblrel. 17, Created)
 DT 01-JUN-2001 (Tremblrel. 17, Last sequence update)
 DT 01-JUN-2001 (Tremblrel. 17, Last annotation update)
 DE MS4A7 PROTEIN.
 OS Mus musculus (Mouse).
 OC Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
 OC Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Mus.
 OX NCBI_TaxID=10090;
 RN [1]
 RP SEQUENCE FROM N.A.
 RA TISSUE-MAMMARY GLAND;
 RA Liang Y., Tedder T.F.,
 RT "Identification of a CD20-, Pepsin/Ribeta-, and Htm4-Related Gene
 RT Family: Sixteen New Ms4a Family Members Expressed in Human and
 RT Mouse.",
 RL Genomics 72:119-127(2001).
 DR EMBL: AF237917; AAK37607.1;
 SO SEQUENCE 234 AA; 25754 MW; CF8B968FB0E033F CRC64;

Query Match 45.8%; Score 44; DB 11; Length 234;
 Best Local Similarity 41.7%; Pred. NO. 10;
 Matches 5; Conservative 7; Mismatches 0; Indels 0; Gaps 0;

OY 4 ISGNLSLFMYRO 15

DB 195 LAGYSSIFMWKQ 206

RESULT 9
 O9L2D5 PRELIMINARY; PRT; 428 AA.
 ID O9L2D5
 AC O9L2D5
 DT 01-OCT-2000 (Tremblrel. 15, Created)
 DT 01-OCT-2000 (Tremblrel. 15, Last sequence update)

DT 01-OCT-2000 (Tremblrel. 15, Last annotation update)
 DE PUTATIVE SECRETED PROTEIN.
 GN SC7A8.23.
 OS Streptomyces coelicolor.
 OC Bacteria; Firmicutes; Actinobacteria; Actinobacteridae;
 OC Actinomycetales; Streptomycetales; Streptomyces.
 OX NCBI_TaxID=1902;
 RN [1]
 RP SEQUENCE FROM N.A.
 RC STRAIN-A3(2);
 RA Oliver K., Harris D.;
 RL Submitted (JAN-2000) to the EMBL/GenBank/DBJ databases.
 RN [2]
 RP SEQUENCE FROM N.A.
 RC STRAIN-A3(2);
 RA Cardeno A.M., Parkhill J., Barrell B.G., Rajandream M.A.;
 RL Submitted (JAN-2000) to the EMBL/GenBank/DBJ databases.
 RN [3]
 RP SEQUENCE FROM N.A.
 RC STRAIN-A3(2);
 RX MEDLINE=97000351; PubMed=8843436;
 RA Redenbach M., Kleiser H.M., Denapate D., Eichner A., Cullum J.,
 RA Kinash H., Hopwood D.A.;
 RT "A set of ordered cosmids and a detailed genetic and physical map for
 RT the 8 Mb Streptomyces coelicolor A3(2) chromosome.",
 RL Mol. Microbiol. 21:77-96(1996).
 DR EMBL: AL137187; CAB69772.1;
 SO SEQUENCE 428 AA; 44436 MW; 11A30F3D74727C38 CRC64;

Query Match 45.8%; Score 44; DB 2; Length 428;
 Best Local Similarity 61.5%; Pred. NO. 19;
 Matches 8; Conservative 1; Mismatches 4; Indels 0; Gaps 0;

OY 3 PISGNLSLFMYRO 15

DB 108 PHAGNGCFWYRO 120

RESULT 10
 O9LPM2 PRELIMINARY; PRT; 556 AA.
 ID O9LPM2
 AC O9LPM2
 DT 01-OCT-2000 (Tremblrel. 15, Created)
 DT 01-OCT-2000 (Tremblrel. 15, Last sequence update)
 DT 01-JUN-2001 (Tremblrel. 17, Last annotation update)
 DE F13K23.13 PROTEIN.
 GN F13K23.13.
 OS Arabidopsis thaliana (Mouse-ear cress).
 OC Eukaryota; Viridiplantae; Streptophyta; Embryophyta; Tracheophyta;
 OC Spermatophyta; Magnoliophyta; eudicotyledons; core eudicots; Rosidae;
 OC eucosids II; Brassicales; Brassicaceae; Arabidopsis.
 OX NCBI_TaxID=3702;
 RN [1]
 RP SEQUENCE FROM N.A.
 RC STRAIN-CV. COLUMBIA;
 RA Liu S.X., Sakano H., Yu G., Lee J.M., Lenz C., Pham P., Tortum M.,
 RA Chin C., Chlou J., Choi E., Chung M., Gonzalez A., Hong B., Liu A.,
 RA Vaysberg M., Altfati H., Brooks S., Buehler E., Chao Q., Conn L.,
 RA Conway A.B., Hansen N.F., Johnson-Hopson C., Khan S., Kim C., Lam B.,
 RA Miranda M., Nguyen M., Palm C.J., Shin P., Southwick A., Davis R.W.,
 RA Ecker J.R., Federspiel N.A., Theologis A.;
 RT "The sequence of BAC F13K23 from Arabidopsis thaliana chromosome 1.",
 RL Submitted (JUN-2000) to the EMBL/GenBank/DBJ databases.
 DR EMBL: AC012187; AAF78503.1;
 DR InterPro: IPR001810; F-box.
 DR Pfam: PF00646; F-box; 1.
 DR SMART: SM00256; FBOX; 1.
 DR PROSITE: PS50181; FBOX; 1.
 SO SEQUENCE 556 AA; 65033 MW; 1D6DB8C95214BF10 CRC64;

Query Match 45.8%; Score 44; DB 10; Length 556;

